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AGRICULTURAL RESEARCH ADMINISTRATION

UNITED STATES DEPARTMENT OF AGRICULTURE

Volume 30 Number 9

September 15, 1946

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THE PLANT DISEASE REPORTER

Series D

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THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station Beltsville, Maryland
Volume 30 September 15, 1946 Number 9

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SCLEROTINIA ROOT ROT OF GUAYULE

W. A. Campbell

Introduction

Sclerotinia sclerotiorum (Lib.) DBy. and S. minor Jagger (2, 3) caused considerable loss of guayule seedlings in the three Emergency Rubber Project nurseries near Salinas, California. The disease was of minor importance in a nursery near San Clemente, California. During the two seasons (1942-43 and 1943-44) that the Salinas nurseries were in full operation, Sclerotinia disease was prevalent in late summer and fall and during the rainy season in late winter and early spring. In one nursery of 153 acres, 1707 spots with sclerotinia-diseased plants were recorded in a roguing operation carried on during the first week in October 1942. Since roguing operations had been carried on since August this represented but a small part of the total loss from Sclerotinia.

Sclerotinia disease was of minor importance in field stands where the plants were spaced 20 to 24 inches apart in rows 28 inches wide except in two irrigated plantings made on areas in which the soil was heavily infested with sclerotia of S. sclerotiorum and S. minor. One of these plantings was in the Salt River Valley near Phoenix, Arizona, on land infested with S. sclerotiorum and the other was near Salinas, on land infested by both species. Out of approximately 30,000 acres of guayule planted by the Emergency Rubber Project, Sclerotinia disease was prevalent only in these two fields comprising an area of approximately 350 acres. In these field plantings the disease developed as a root rot from underground mycelium without above-ground infections, contrary to the condition observed in the coastal nurseries.

Description and History of Infested Field

The studies on Sclerotinia root rot herein reported were conducted on a 256-acre irrigated field on the Jacks Ranch leased by the Emergency Rubber Project for a guayule plantation. This field is approximately 5 miles southeast of Salinas and is situated on flat gently sloping bottomland flanked on two sides by banks 25 to 30 feet high. The soil on 200 acres was classified as a silt loam and that on 56 acres varied from a silty clay loam to silty clay. Although the land had been leveled for irrigation, irregularities existed and areas of varying size were poorly drained.

Prior to 1942 the field had been used for lettuce and other crops grown under irrigation. For several years lettuce alone was grown,

sometimes as many as three crops in one year. During this period of intensive lettuce culture losses from Sclerotinia became increasingly more severe.

The 256-acres in the Sclerotinia-infested area was divided into two fields of approximately equal size for administrative purposes. Field C which comprised the northern half was planted to guayule in November and December 1942. This area had been planted to radishes and beans the previous season. Because of a lack of nursery stock the southern half or Field D was subleased during the summer of 1943 and planted to lettuce and black valentine beans with sunflower windbreaks. By September 1, 1943, 90 percent of the sunflowers had succumbed to Sclerotinia rot (mainly caused by S. minor) (1). Considerable disease was also present in the beans which had formed a dense mass of vegetation on the ground, and a small number of infected lettuce plants was observed. Most of the disease in the beans, judging by the size of the sclerotia, was caused by S. sclerotiorum, but that in the lettuce was chiefly S. minor.

Field D was planted to guayule from November 1943 to January 1944. Twenty-two acres planted in November 1943 were replanted in May 1944 owing to poor survival of the first planting.

The amount of irrigation water applied to the guayule plantings determined to a large degree the loss from Sclerotinia disease. Field C was irrigated 3 times in 1943, during May, July, and October, with a total application of 20.4 acre inches of water. In 1944 no irrigation was applied to Field C and only enough to Field D to insure establishment of the stand. Exact figures were not obtained for 1945, but both fields were heavily irrigated throughout the growing season.

Extent of Loss

Guayule transplants killed by Sclerotinia root rot were first observed by Braun (1) in Field C of the Jacks Ranch early in May 1943 when the first survival count was made. Continued observations and surveys throughout the summer demonstrated that new infections continued to develop as long as sufficient moisture for the growth of Sclerotinia was present in the soil. Inasmuch as the 1943 irrigations were comparatively light the periods favorable for fungus development were relatively short and the total loss throughout the season did not exceed 1 percent.

Losses during 1944 were very light in both Fields C and D and were observed only in the spring. With a change of policy in 1945, heavy irrigations were given throughout the summer months in order to stimulate shrub growth. These irrigations were started early in the spring and continued until late in the season. Plants killed by Sclerotinia root rot were first noticed when temperatures became favorable for growth.

Diseased plants failed to break dormancy and gradually died, or lush plants wilted suddenly when the roots were attacked. Systematic surveys for disease loss were made in May for Field D and in August for Field C. The results are presented in Table 1. Since further dying occurred after the surveys were made the indicated loss in Field D is less than the actual loss because dying plants were observed as late as September. The loss from Sclerotinia root rot was not evenly distributed over the field and was more severe in some areas than others. In general, losses were an index to the topography of the field inasmuch as the heaviest losses occurred in flat poorly drained areas.

Table 1. Losses from Sclerotinia disease in a 256-acre guayule planting near Salinas, Calif.

Date survey	Age of shrub at time of survey	Approximate area	Plants diseased
	Months	Acres	Percent
May 1945	17	29	13.4
May 1945	12	22	10.3
May 1945	17	39	7.8
May 1945	16	38	6.2
August 1945	32	32	6.1
August 1945	32	32	14.4
August 1945	32	32	5.3
August 1945	32	32	8.4

Sclerotinia spp. Causing Loss

Isolations made from diseased plants from Field C in 1943 and from Fields C and D in 1944 indicated that both Sclerotinia sclerotiorum and S. minor were causing infections. However, S. minor was isolated from most of the plants examined. Random examination of diseased plants in the course of the survey of Field D in May 1945 disclosed that most of the rotted roots were covered with sclerotia of the S. minor size. In order to determine the relative number of plants killed by the two species of Sclerotinia, random selections of recently killed or wilting plants were made in July and August 1945 in both the 18 and 32 months old shrub. The type of sclerotia present on the tap root of each plant was recorded. In addition, the location of the lesion was noted and isolations were made from diseased tissue of each plant. The results as given in Table 2 show that for Fields C and D, S. minor was responsible for 83 percent of the infections and S. sclerotiorum for 17 per-

cent. S. minor infections were somewhat more numerous in Field C where the shrub was 32 months old, accounting for 87 percent of the infections. In Field D where the shrub was 18 months old 79 percent of the infections were caused by S. minor.

Sclerotinia sclerotiorum proved to be more easily isolated from diseased roots than S. minor. Twenty-seven out of 28 roots on which sclerotia of the S. sclerotiorum type were present yielded the organism in culture. From 134 roots with sclerotia of the S. minor type, 105 yielded pure cultures of the fungus. The remaining roots yielded species of Fusarium, Penicillium, and Rhizopus.

Table 2. Type of sclerotia present on roots and species of Sclerotinia isolated from diseased plants selected at random in guayule planting.

Approximate age of plants	Total plants[a]	S. sclerotiorum: Plants with sclerotia		Isolated	S. minor: Plants with sclerotia		Isolated
Months	Number	Number	Percent	Number	Number	Percent	Number
18	90	19	21	18	71	79	48
32	72	9	12	9	63	87	57
All	162	28	17	27	134	83	105

[a] Includes only diseased plants having sclerotia on or in root lesions.

Location of Root Lesions

An important difference between Sclerotinia minor and S. sclerotiorum is the manner in which they resume vegetative growth after weathering unfavorable environmental conditions as sclerotia. The sclerotia of S. minor germinate invariably to produce a mycelium that attacks the roots or other parts of susceptible host plants. The sclerotia of S. sclerotiorum on the other hand, usually develop apothecia and the vegetative stage is resumed when the ascospores produced by these germinate on dead or weakened host tissue. The apothecial stage of S. minor was obtained under experimental conditions but was not observed in the field.

Observations on diseased plants in the Jacks field indicated that practically all infections occurred below the soil surface. Throughout most of the season, with the exception of the rainy period in the winter and spring or immediately following irrigations, cultivations for weed control produced a loose soil cover between the rows. The

surface soil, not only between the rows but in the rows, was ordinarily too dry for apothecial formation down to a depth of 2 or 3 inches. In order to determine whether or not ascospore infection by S. sclerotiorum was playing any important part in field infections, the location and extent of all lesions were noted on the plants pulled for isolation purposes.

Most of the lesions noted on the plants involved all of the root and no definite point or origin was evident (Table 4). However the root lesions usually stopped an inch or so below the crown where the fungus was checked by drying, which was considered evidence that they had originated lower in the soil. Forty-six S. minor lesions were confined to the lower or middle root and 19 involved the crown or upper root. Twelve S. sclerotiorum lesions were limited to the lower or middle root and only 6 were at the root crown or upper root. These 6 could possibly have originated as ascospore infections, inasmuch as dead leaves suitable for the saprophytic development of these spores were present at the crown in contact with the ground.

Table 4. Location of lesions on the tap roots of sclerotinia-diseased guayule.

Fungus Species	Approx. age of plants Months	Total plants[a] examined Number	Location of lesion on tap root: Upper root Number	Middle root Number	Lower root Number	Whole root diseased[b] Number
S. minor	18	76	11	4	15	46
	32	67	8	0	27	32
S. sclerotiorum	18	21	4	2	5	10
	32	12	2	0	5	5
Both species		176	25	6	52	93

[a] Includes plants not reported in Table 3.
b Rot so far advanced that the origin of the lesion could not be determined.

Sclerotial Population of the Soil in Relation to Root Rot

If all Sclerotinia minor and most S. sclerotiorum infections originated from soil mycelium presumably produced by sclerotia, the number of sclerotia in the soil should be related to the severity of disease loss. In order to determine the relative numbers of sclerotia in areas with varying disease loss 14 plots were established, 3 in Field C and

11 in Field D. The number of diseased plants per plot varied from practically none to over 60 percent of the existing stand (Table 5). Each plot was 20 plants long and 10 rows wide. The number of living and dead plants per row were recorded for each plot. By means of random numbers 8 locations were selected in each row for a soil sample. Four samples were taken in the row itself and 4 in the space between the plants. The soil samples were taken with a soil tube to depth of 6 inches. The total surface area of the 80 samples taken in a plot was equivalent to 1 sq. foot.

The 80 samples from each plot were mixed together and washed through 2 screens, following the procedure used by Leach and Davy (4) to recover sclerotia of Sclerotium rolfsii Sacc. The first screen was of 14-mesh galvanized wire and the second of 20-mesh stainless steel wire. The larger sclerotia were stopped on the 14-mesh screen and could readily be recognized. The smaller ones were more difficult to find and necessitated examination of the screen with a reading glass. Verification of sclerotia was made with the aid of a binocular dissecting microscope.

The number of sclerotia recovered per square foot per plot ranged from 30 to 121 (table 5). In viability tests made by surface sterilizing the sclerotia with 30 percent hydrogen peroxide for 3 minutes and plating on Sleeth's medium (5) from 88 to 100 percent of the sclerotia germinated. The distribution of sclerotia by Sclerotinia spp. showed considerable variation. In all but 2 of the 9 plots from which sclerotia were plated, more sclerotia of S. sclerotiorum than of S. minor were recovered by soil sampling. This is in direct contradiction to the presence of disease in the field, since only 17 percent of the loss was caused by S. sclerotiorum (Table 2).

Comparison of the percentage loss per plot with the number of sclerotia recovered per square foot revealed no clearcut relationship. Apparently sclerotia were distributed throughout the soil in both Field C and D in considerable numbers. Soil moisture was probably the determining factor in disease expression, considering that sufficient viable sclerotia were present in each plot to cause infection. The plots with the greatest disease loss, that is 12 and 13, were in an area which because of its topography received much more water than surrounding areas where disease loss was small. Plot 14 with the smallest disease loss but with a large sclerotial population was in a high spot close to plots 12 and 13 and consequently received less water. Likewise in the 18-month-old shrub disease loss was more closely correlated with moisture than with the number of sclerotia of either S. minor or S. sclerotiorum present in the soil.

Table 5. Sclerotial population of the soil in relation to the number of diseased plants, viability of sclerotia, and distribution according to Sclerotinia spp.

Plot	Approx. age of plants	Total plants	Plants dead	Sclerotia per sq. foot	Viability of sclerotia	Distribution of species	
	Months	Number	Percent	Number	Percent	S. minor Percent	S. sclerotiorum Percent
1	18	168	8	37	--	--	--
2	18	107	20	55	100	35	65
3	18	175	17	44	94	35	65
4	18	164	18	67	100	39	61
5	18	148	22	59	--	--	--
6	18	114	27	43	88	41	59
7	18	168	20	71	100	46	54
8	18	156	5	51	95	20	80
9	18	154	25	30	96	65	35
10	18	168	11	41	--	--	--
11	18	157	5	40	90	50	50
12	32	132	46	121	96	6	94
13	32	143	62	110	--	--	--
14	32	134	.7	109	--	--	--

Summary

Root rot caused by Sclerotinia minor and S. sclerotiorum was responsible for considerable loss in a 256-acre guayule planting near Salinas, California over a 3-year-period. The disease developed as a root rot with little evidence of above-ground infection by ascospores.

In 1943 under moderate irrigation the loss was less than 1 percent; in 1944 when no irrigation water was applied very few plants died; however, in 1945 under heavy summer irrigation the average loss was 9 percent with a range of 5.3 to 14.4 percent for different areas.

As determined by isolations from diseased plants selected at random 83 percent of the loss was caused by S. minor and 17 percent by S. sclerotiorum.

In soil samples taken at random from 14 plots the sclerotial population of the soil was found to range from 30 to 121 per square foot. From 88 to 100 percent of the sclerotia from 9 plots were viable when surface sterilized and plated on nutrient agar. In all but 2 of the 9 plots more sclerotia of S. sclerotiorum than S. minor were recovered. Disease loss was not related to the sclerotial population.

The amount of loss was closely related to soil moisture which in turn was influenced by rain, irrigations, and topography. Soil temperatures were favorable for infection from March to November and moisture was the limiting factor throughout most of the year. Cultivations and low humidity during the growing season dried out the soil surface restricting the activity of the fungi to soil layers below 2 or 3 inches.

Literature Cited.

1. Braun, A. J. Observations on Sclerotinia root rot of guayule. U.S. Dept. Agr. Plant Dis. Reptr. 28: 982-984. 1944.
2. Campbell, W. A., L. D. Leach, J. T. Presley, and W. C. Snyder. Some diseases of guayule in California. U.S.Dept. Agr. Plant Dis. Reptr. 27: 63-66. 1943.
3. _____, and J. T. Presley. Diseases of cultivated guayule and their control. U.S.Dept. Agr. Circ. 749. 1946.
4. Leach, L. D., and A. E. Davey. Determining the sclerotial population of Sclerotium rolfsii by soil analysis and predicting losses of sugar beets on the basis of these analyses. Jour. Agr. Res. 56: 619-631. 1938.
5. Sleeth, B. Agar medium and technique for isolating Pythium free of bacteria. Phytopathology 35: 1030-1031. 1945.

SPECIAL GUAYULE RESEARCH PROJECT
SALINAS, CALIFORNIA

EASTERN WHITE PINE AFFECTED WITH NEEDLE BLIGHT IN MARYLAND

E. A. Walker

Needle blight (physiogenic) made its appearance in Maryland early in July. Previously it had been of little concern to nurserymen and growers of ornamentals. The disease has been most severe in the Rockville and Sugarloaf Mountain areas of Montgomery County. Isolated cases have been observed to a limited extent on young and old trees in Frederick, Carroll, and Prince George's Counties.

Needle blight affects the current season's growth of needles, causing them to turn reddish-brown in color and to die rapidly from the tip back one-fourth to one-half the length of the needle. The affected tree appears from a distance to be dying as if scorched by fire. Severely affected trees will be stunted in growth for three to five years, and an occasional tree may die. As observed this year, needle blight does not affect all trees in a white pine planting. One tree in a group may be severely affected, and another nearby may show only slight coloration, while the remaining trees appear healthy.

Needle blight is thought to be caused by a combination of unfavorable weather and root growth. Spaulding and Hansbrough[1] mention that the condition appears after prolonged periods of cloudy, moist weather, followed abruptly by dry, hot days. This is associated with the dying of feeder rootlets, resulting in less water being taken up to supply the young needles which are then scorched by the heat. This explanation fits the situation in Maryland this year. The hot, dry weather apparently came when the needles were most tender, in the area where needle blight is worst.

Application of nutrients in the form of commercial fertilizer this fall followed by heavy watering and then mulching with an abundant supply of pine needles is recommended as the best treatment to stimulate new feeder root development for the best growth response of affected trees.

UNIVERSITY OF MARYLAND
JULY 24, 1946

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[1] Spaulding, Perley and J. R. Hansbrough. The needle blight of eastern white pine. (Mimeographed). US Dept. Agr. 1943.

RATE OF SPREAD OF BLUEBERRY STUNT IN NORTH CAROLINA

J. B. Demaree

Blueberry stunt has been known in New Jersey for several years and was reported by Wilcox in 1942 as being a virus disease[1]. Its rate of spread in New Jersey blueberry fields at first was insignificant and excited little or no alarm. During the past 10 years there has been a slow but constant increase of infected bushes in some fields until at present production in a few of the earlier affected plantings has been so low as to be unprofitable.

The disease is now known in several fields in New Jersey, in a few fields in North Carolina, and a few infected bushes have been seen in each of the States of Massachusetts, Michigan, and New York.

This virus disease seriously affects the berry-producing capacity of infected blueberry bushes. While the canes of field-grown highbush blueberries (Vaccinium australe Small) normally grow to the height of 4-6 feet, the basal shoots of infected bushes seldom attain the height of 18 inches; consequently, the older bearing canes or shoots cannot be replaced by vigorous new shoots. When all canes of an infected bush are cut off at the ground surface, a new bush of normal height is never formed.

The bearing wood of a recently infected mature bush is reduced by this stunting of the bearing shoots. Moderately infected bushes blossom and set some fruits but the berries are small, and of an inferior quality, and when ripening adhere tightly to the peduncles; consequently, infected bushes are not picked. Stunt finally makes the bushes barren.

The disease is known in 6 North Carolina blueberry plantings (1945). Cultivation of blueberries in this State is a relatively new industry; likewise, the stunt disease has not been known there much beyond the present decade. Even so, 2 of the 6 infected fields have become so unproductive during the past 5 years on account of the disease that parts of them have already been abandoned.

The rate of spread of blueberry stunt varies in different fields and in different sections or regions. Its natural rate of spread is moderate to rapid in New Jersey, very rapid in some fields in North Carolina, and slow or nil in Massachusetts, Michigan, and New York. The determining factor influencing the rate of dissemination in fields may be associated with the presence and abundance of a vector. Circum-

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[1] Blueberry stunt, a virus disease. R. B. Wilcox. Plant Dis. Reptr. 29: 211-213. May 1942.

stantial evidence suggests that initial infections in new and isolated plantings originate from propagating stock introduced from an infected field or region rather than from viruliferous insects migrating from wild blueberry or other Ericaceous plants in nearby wastelands. It is assumed that an insect vector is present in New Jersey and in North Carolina, but absent in blueberry fields in Massachusetts, Michigan, and New York, and that the few diseased bushes observed in the 3 northern States were infected when introduced as propagating stock from some other region.

While the writer had observed the increase of stunt in blueberry fields in New Jersey and North Carolina for several years, no information was available as to the probable productive life of a planting after the disease was once established.

This report describes the rate of spread of stunt in one blueberry field planted in 1936 near Magnolia, North Carolina. This field was planted adjacent to an older field where the disease had become established earlier. The accompanying chart (fig. 1) shows how the two fields border each other and the relationship of the probable source of infection to the new field.

Field 1 (old field) was established about 1928 from plants propagated from the owner's fields in New Jersey, which at that time were relatively free of the stunt disease. Field 2 (new field) was set with plants also propagated and grown from the owner's field. The two fields are separated by a narrow roadway and an open drainage ditch. The east end of the two fields is bounded by a highway. Across the highway to the east is another blueberry field, but younger than the other two. South of field 1 is a wooded area with some Ericaceous plants, including wild highbush blueberry. North of field 2 is an almost impenetrable growth of swamp plants, including Ericaceous species. An open field lies to the west.

The blueberry bushes in the east third of field 1 have made an irregular, uneven, and otherwise unsatisfactory growth, at least since 1935. Some rows made a fairly good growth, while others grew slowly and were unproductive. The cause of the unhealthy condition of the bushes was at first unknown, but was suspected of being due to a high water table, deficiency of soil nutrients, etc. In 1938 the cane canker disease, caused by Physalospora corticis Demaree and Wilcox, was found causing severe damage to the canker-susceptible varieties. About this time it was suspected that stunt was also involved in the disease complex. Stunt at that time was known only in New Jersey, and its virus nature had not yet been demonstrated. By 1940 stunt was definitely identified in the east end of the old field and was found to have been well established. It is now suspected that a few of the originally set plants were infected with stunt and that it gradually spread to the bushes in that area. Finding both canker and stunt here

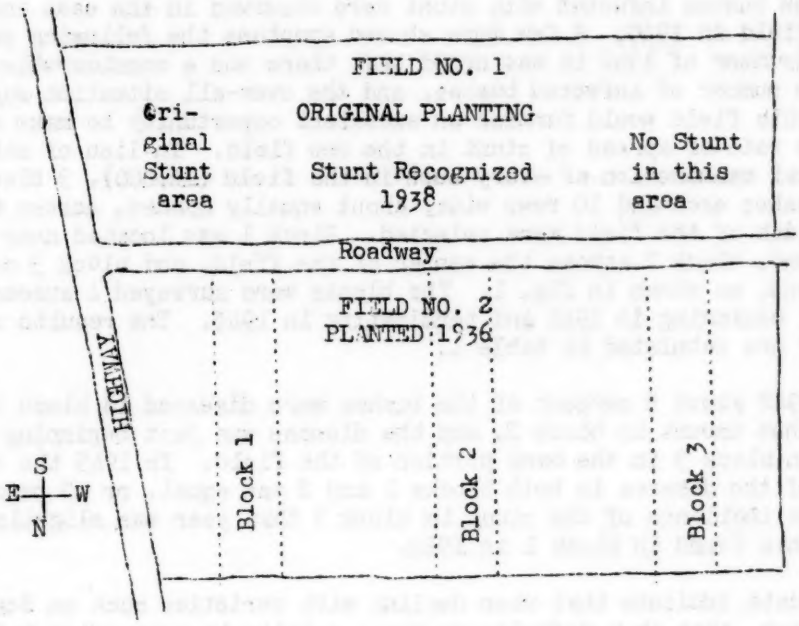

Figure 1. Plan of a North Carolina planting consisting of two adjacent fields showing the position of the 3 blocks in which records were taken on the spread of the stunt virus disease.

furnished an explanation for the slow and abnormal growth of bushes observed for several years in the east end of field 1. It also gave a clue as to why one or two varieties grew more normally; that is, they were resistant to both canker and stunt.

Stunt has never been observed in the west end of field 1. This is not easily explained. We do know, however, that the two very susceptible varieties, Scammell and Cabot, were not planted in that end. Of course, it is possible that no infected plants were set there originally.

Field 2 was planted to 4 varieties, Cabot, June, Scammell, and Rancocas; with Cabot and Scammell predominating. Both these latter

varieties are quite susceptible to stunt while Cabot is also very susceptible to canker. June is moderately susceptible to stunt, and Rancocas, if susceptible, shows very slight if any stunt symptoms.

A few bushes infected with stunt were observed in the east end of this field in 1940. A few more showed symptoms the following year. In midsummer of 1942 it was noted that there was a considerable increase in the number of infected bushes, and the over-all situation suggested that this field would furnish an excellent opportunity to make a study of the rate of spread of stunt in the new field. In lieu of making a critical examination of every bush in the field (14,000), 3 blocks of 600 bushes each and 10 rows wide, about equally spaced, across the narrow width of the field were selected. Block 1 was located near the east end, block 2 across the center of the field, and block 3 near the west end, as shown in fig. 1. The blocks were surveyed 4 successive years, beginning in 1942 and terminating in 1945. The results of the survey are tabulated in table 1.

In 1942 about 9 percent of the bushes were diseased in block 1, about half that amount in block 2, and the disease was just beginning to appear in block 3 in the back portion of the field. In 1945 the concentration of the disease in both blocks 1 and 2 was equal, or 47 percent, and the incidence of the stunt in block 3 that year was slightly less than that found in block 1 in 1942.

The data indicate that when dealing with varieties such as Scammell and Cabot, that show definite symptoms relatively soon after infection (and assuming the presence of a vector and a few foci of infection), the spread of stunt in North Carolina fields may be extremely rapid among bushes within a short radius, but relatively slow to distant uninfected or slightly infected areas of a field. This may indicate that the vector is active within a narrow range but does not migrate far from its habitat.

Table 1. Rate of spread of blueberry stunt in 3 blocks in a North Carolina blueberry field from 1942 to 1945, inclusive.

	Block 1				Block 2				Block 3			
Year	Apparently healthy bushes	New infection	Accumulative infected bushes No.	%	Apparently healthy bushes	New infection	Accumulative infected bushes No.	%	Apparently healthy bushes	New infection	Accumulative infected bushes No.	%
1942[a]	545	55	55	9.0	572	28	28	4.7	596	4	4	.66
1943	460	85	140	23.3	518	54	82	13.7	591	5	9	1.5
1944	409	51	191	31.7	458	60	142	23.7	580	11	20	3.33
1945	318	91	282	47.0	318	140	282	47.0	553	27	47	7.8

[a] G. A. Meckstroth, United States Department of Agriculture, recorded the data for 1942.

Assuming that the surveys made in this stunt-infected blueberry field near Magnolia, North Carolina, reflect a true criterion as to the normal spread of the disease in southern fields, then it may be predicted that the expected profitable productive life of a slightly stunt-infected field in that region may not be over 5 years. It may also be inferred that by roguing affected plants or removing affected and adjacent plants the disease may be prevented from spreading rapidly to other parts of a large field.

Summary

Stunt, a virus disease of blueberries, spreads rapidly after it becomes established in North Carolina and New Jersey blueberry plantings, but that spread is either slow or lacking in Massachusetts, Michigan, and New York. The rate of dissemination in fields may be governed by the presence, activity, and abundance of an insect vector. Circumstantial evidence suggests that initial infections in new and isolated plantings originate from diseased propagating stock introduced from an infected field rather than from viruliferous insects migrating from wild blueberry plants in nearby wastelands.

A record of the spread of the disease was made in a 10-acre blueberry field in North Carolina from 1942 to 1945, which showed an increase of diseased plants in one block from 4.7 percent in 1942 to 47 percent in 1945.

DIVISION OF FRUIT AND VEGETABLE CROPS AND DISEASES

AN APPARENT RELATIONSHIP BETWEEN SOIL MOISTURE AND BORDEAUX INJURY TO ALMOND FOLIAGE

E. E. Wilson

Bordeaux mixture and other copper fungicides sometimes injure almond foliage within a few weeks after the post-blossom (leaf-cover) spray is applied. This injury is manifested as dark brown to almost black necrotic areas on the tips and along the margins of the distal half of the leaf. Brown to reddish flecks also develop over other areas of the leaf blade. Some yellowing and dropping of leaves may occur.

A type of bordeaux injury, in which extensive scorching of the leaf blade does not occur, may develop two months or more after the post-blossom spray. Leaves will lose their green color, become yellow, and fall during or shortly after a period of high temperatures. Most frequently a few of the older leaves only are involved and consequently defoliation is not severe. In one orchard, however, defoliation became so severe in 1946 as to cause considerable shriveling of the nuts. This orchard, located on relatively shallow soil of low water-holding capacity, was not irrigated in early summer. By mid-June the leaves of non-sprayed trees would wilt slightly in mid-day but recover during the night. Leaves on sprayed trees, on the other hand, wilted much more severely and many of the older leaves became yellow and fell. Upon application of water to the soil the foliage of such trees promptly regained a turgid condition, and the yellowing and dropping of leaves was checked. Sprayed trees located in orchards that received an early-summer irrigation bore no recognizable signs of this type of injury. None of the scorching type of injury was observed in 1946.

Trees of the Ne Plus Ultra variety suffered most from the yellow-leaf type of injury, but Nonpareil and Drake trees were also affected. Ne Plus Ultra also appears to be the variety most subject to the scorching type of injury.

DIVISION OF PLANT PATHOLOGY
UNIVERSITY OF CALIFORNIA
DAVIS, CALIFORNIA

DILOPHOSPORA ALOPECURI ON WHEAT IN SOUTH CAROLINA

A. G. Johnson and R. W. Leukel

Specimens of wheat heads infected by Dilophospora alopecuri (Fr.) Fr. along with a specimen of threshed wheat infested with wheat nematode galls were received July 23, 1946, from E. W. Montgomery of Greenville, South Carolina. This is believed to be the first report of the occurrence of D. alopecuri on wheat in the United States. The nematode galls amounted to 21 percent of the wheat sample (by count) but the severity of the fungus disease could not be determined from the small specimen received, nor was this stated in the letter.

In the United States, D. alopecuri has been reported previously on various grasses and also on rye as follows:

Host	Where found	Year Collected	Collector
Agropyron smithii	Utah	1925	A. O. Garrett
" spicatum	Washington	1937	G. W. Fischer
" trachycaulum	North Dakota	1941	R. Sprague
" "	Wyoming	1941	J. R. Hardison and G. W. Fischer
Calamagrostis canadensis	Wisconsin	1905	J. J. Davis
Holcus lanatus	California	1923	G. H. Godfrey
" "	Oregon	1915	H. S. Jackson
" "	"	1937	G. W. Fischer
" "	"	1937	R. Sprague
" "	"	1938	" "
" "	"	1939	" "
Melica bulbosa	Wyoming	1941	G. W. Fischer
Poa sp.	Utah	1940	G. W. Fischer and Raeder
Secale cereale	Oregon	1944	C. C. Anderson
Sitanion jubatum	Washington	1937	R. Sprague
" "	"	1941	G. W. Fischer

In Europe, the disease has been reported on various cereals and grasses in England, Norway, Denmark, Holland, France, Belgium, Switzerland, Germany, and Serbia. Atanasoff (Phytopathology 15: 11-40. 1925) reported that the infection of wheat, spelt and rye by Dilophospora alopecuri occurred only in association with the wheat nematode (Anguina tritici) and that no infection occurred in the absence of the nematode.

DIVISION OF CEREAL CROPS AND DISEASES

NEW OAT DISEASE IS WIDESPREAD IN ILLINOIS IN 1946[1]

L. R. Tehon and G. H. Boewe

A survey of oat fields, made this year, has shown that a new disease, Helminthosporium blight,[2] is present over most of Illinois and was very destructive this year in some fields of susceptible varieties. Helminthosporium blight is a seed-borne, fungous disease which attacks the plant both in the seedling stage and in later stages when it infects the nodes.

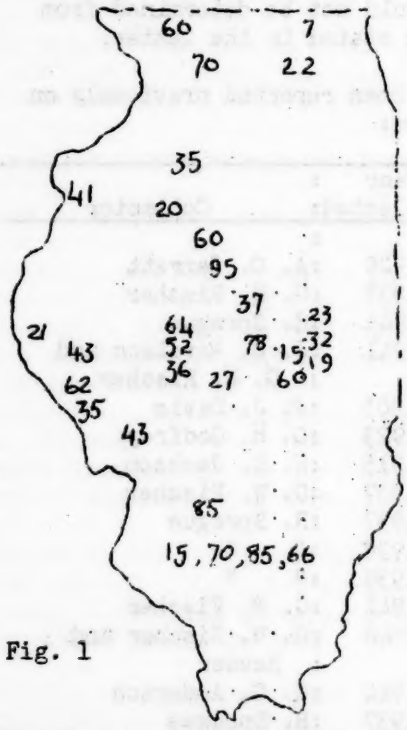

Fig. 1

Percent of oat plants infected with Helminthosporium blight.

Fig. 2

Estimated severity of seedling blight.

This disease was found in varying amounts in fields of Tama, Vicland,

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[1] From mimeographed publication of Illinois Natural History Survey, addressed to Illinois Farm Advisers, July 26, 1946.

[2] Meehan, Frances, and H. C. Murphy. A new Helminthosporium disease of oats. Abstr. Phytopathology 26: 406. 1946.]

and Boone varieties. Figure 1 shows the percent of plants found infected at the time field counts were made (June 4 to July 17), and the approximate location in the State of the fields examined.

Losses caused by Helminthosporium blight vary from field to field, depending upon the amount of seedling blight and the stage of development of the plant when node infection occurred. Reduction in yield was slight in many fields, but some fields were almost complete failures. Many growers report fair yields but low test weights. In several cases the old variety Columbia, which is highly resistant, outyielded Tama or Vicland when grown in the same field.

The seedling phase of Helminthosporium blight was severe last spring in some localities. Figure 2 shows estimates of the severity of this phase of the disease made during our survey. Seedling blight was most severe in the southwest. In this region, few fields were plowed up and planted to another crop because of the severe reduction in stand due to seedling blight.

Since the spores of the fungus are carried on the seed, all seed oats should be treated. According to Dr. H. C. Murphy of the United States Department of Agriculture at Ames, Iowa, the seedling phase of the disease can be effectively controlled by treating the seed with New Improved Ceresan. Seed treatment will not prevent joint infection. However, it is quite possible that there will be less joint infection in fields where treated seed is sown.

In addition to seed treatment, good farm practices such as crop rotation and the use of cleaned seed will help in reducing the amount of infection in next year's crop. Fortunately, there are certain varieties of oats that are resistant to the disease. According to Dr. Murphy[3], all Bond-hybrid selections, including Clinton and Benton, and such older varieties as Marion, Iogold, Erban and Richland are resistant. Vicland, Boone, Tama, Fultex, Osage, Cedar and Neosho are susceptible. No Helminthosporium blight has been observed on Columbia.

In the field, leaves of infected seedlings have a peculiar green color and a reddish brown stripe 1 to 3 mm wide which runs along one edge of the leaf for the entire length of the blade. Examination of the diseased seedlings shows various stages of rot on the root system, scutellum and even the crown or first node. Severely diseased seedlings die. Less severely diseased seedlings may die at any later stage of development or live to maturity, in which case they are usually weak plants.

[3 Murphy, H. C., and Frances Meehan. Reaction of oat varieties to a new species of Helminthosporium. Abstr. Phytopathology 36: 407. 1946.]

In older plants the first sign of infection is a brownish translucence of the infected node followed by a light brown color of the leaf sheath above the affected node. Leaves on diseased plants develop reddish brown stripes in the blades, beginning with the lowest leaf and developing progressively up the plant. Later the affected nodes become black, due to the development of fruiting structures of the fungus. Plants with early infection ripen prematurely. Straw above the diseased nodes is weak and brittle; it breaks over very easily just above the infected joint. A badly diseased field has the appearance of a wheat field with severe fly infestation.

ILLINOIS STATE NATURAL HISTORY SURVEY
URBANA, July 26, 1946

NORTHERN ANTHRACNOSE OF RED CLOVER IN ILLINOIS IN 1946

G. H. Boewe

A survey of red clover fields in Illinois (May 21-29) has shown that northern anthracnose caused by Kabatiella caulivora (Kirchn.) Karak., one of two important anthracnose diseases of red clover, is widespread and very destructive in some fields. In central Illinois the disease was very severe in many fields, it was much less severe in the northern part of the State, and very light in the southern third. (Figure 2).

Losses due to northern anthracnose varied from field to field, ranging from a trace to 75 percent of the hay crop. Reduction in hay yield will probably average 25 percent for the entire central part of the State. In the northern part, losses will not be very great in most fields.

Northern anthracnose is a cool, wet weather disease. In ordinary years it causes very little damage in Illinois clover fields. But when weather conditions are favorable for its development, as they were in some sections of the State in the springs of 1945 and 1946, northern

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Legend to Figure 1.

1 = Central Illinois: Average of mean temperatures and total rainfall recorded at Decatur and Urbana.

2 = Northern Illinois: Average of mean temperatures and total rainfall recorded at Marengo and Scycamore.

3 = Southern Illinois: Average of mean temperatures and total rainfall recorded at Flora and Carlyle.

S = Severe outbreak of anthracnose.
L = Light attack of anthracnose.

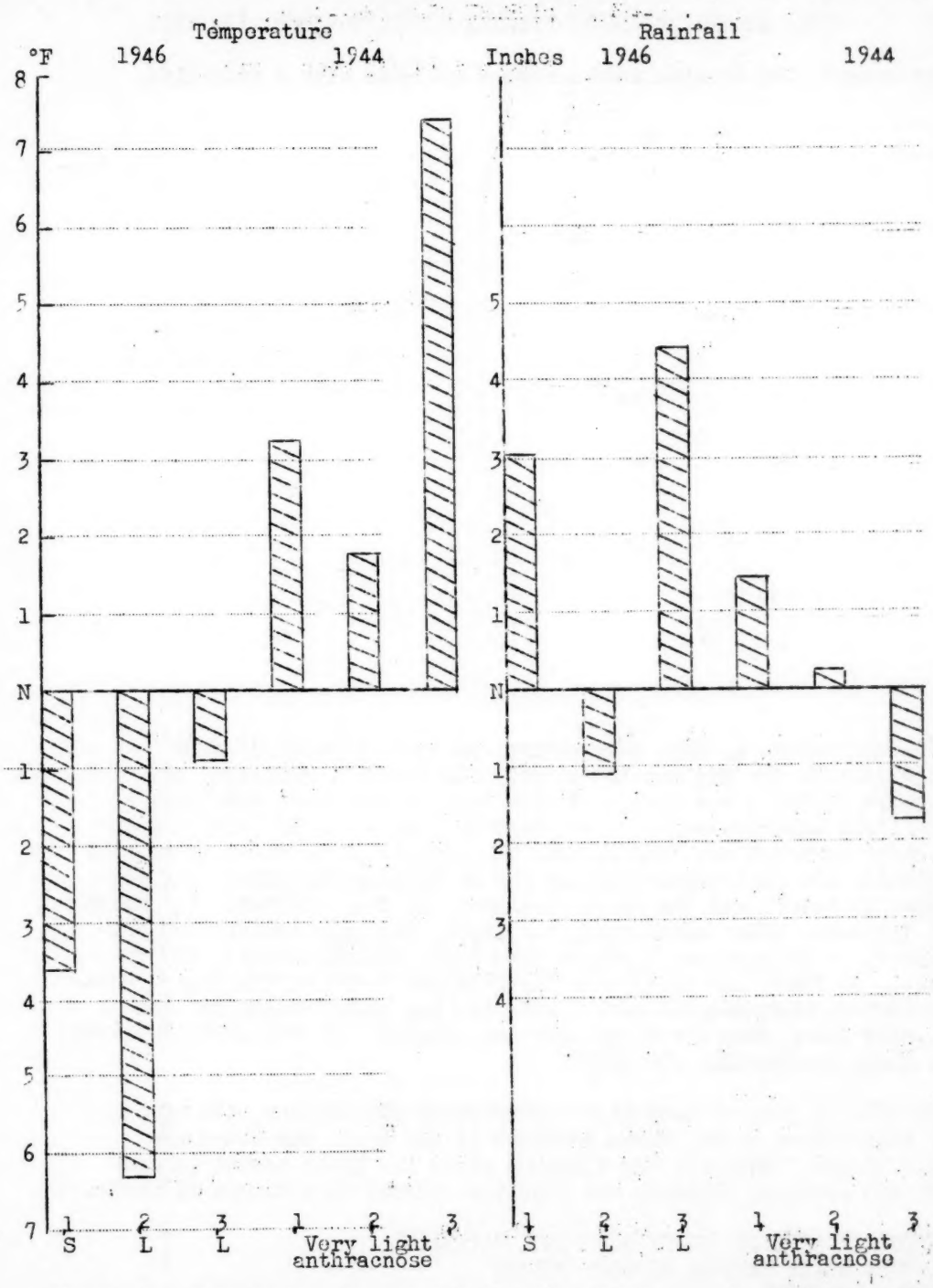


Figure 1. Departures above and below the State normals of May temperature and rainfall in three sections of Illinois during May, 1946, and May, 1944 (See opposite page for symbols)

anthracnose will develop into a severe epidemic over a wide area.

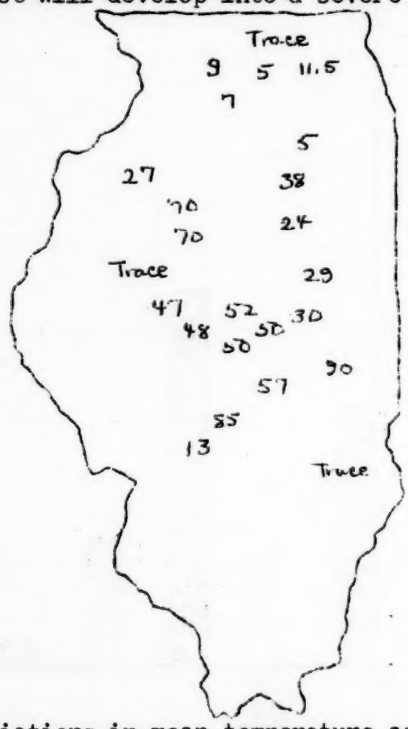


Fig. 2. Percent of red clover stems infected by northern anthracnose in Illinois, 1946.

The deviations in mean temperature and average rainfall from the State normals for May for two stations in central, northern, and southern Illinois for 1946, a severe anthracnose year, and 1944, a very light anthracnose year, are shown in Figure 1. In 1946, a year in which northern anthracnose reached epidemic proportions in central Illinois, the mean temperature of May at Decatur and Urbana was considerably below, and the rainfall above, the State normal. In northern Illinois, where anthracnose was light, the mean temperature and rainfall at Marengo and Sycamore were below the May normals for the State. At Flora and Carlyle in the southern third of the State, where northern anthracnose was very light, the May mean temperature was slightly lower than the State mean and rainfall was considerably above the State average for the month.

In 1944, a year of very light anthracnose development, the mean May temperature in all three sections of the State was above the State normal. Rainfall was slightly above the State normal in central and northern Illinois and less than normal in southern Illinois.

SECTION OF APPLIED BOTANY AND PLANT PATHOLOGY
ILLINOIS STATE NATURAL HISTORY SURVEY
URBANA, ILLINOIS

BRIEF NOTES ON DISEASES OF CEREAL AND FORAGE CROPS

A CORRECTION: STALK ROT OF CORN AND SORGHUM

By E. C. Tullis

In an earlier note in the Plant Disease Reporter (PDR 28 (30): 1100) it was reported that Spicaria elegans var. sorghina had been found on corn in the eastern part of Texas, and on sorghum in north and west Texas. The fungus has been identified by Dr. C. D. Sherbakoff as the microconidial stage of Fusarium moniliforme Sheldon. The previous identification was therefore incorrect. -- Division of Cereal Crops and Diseases, U.S. Bureau of Plant Industry, Soils, and Agr. Engineering, Agricultural Research Administration, U.S. Department of Agriculture, Beaumont, Texas.

SOYBEAN LEAF SPOTS IN MARYLAND

By E. A. Walker

Cercospora leaf spot of soybean (Cercospora cruenta ?) was found abundant on the leaves of the Black Wilson variety in an eight-acre field near Harmony, Caroline County, on July 22. The spots were small, ranging from pinhead to 3 mm in size, apparently developing from a very recent infection. The right-hand leaflets on 20 leaves collected at random were used to count the number of leaf spots. Counts averaged 18.5, and ranged from 4 to 72 spots per leaflet. This field showed the heaviest Cercospora leaf spot of any field ever examined in the past three years.

Phyllosticta leaf spot (Pleosphaerulina sojaecola) was found associated with Cercospora leaf spot in the same field near Harmony. This disease causes a firing of the leaflet margins, which turn brown and may drop away from the rest of the leaflet. In the dead tissue abundant black fruiting bodies of the pycnidial (Phyllosticta sojaecola) stage of the fungus are produced. Considerable leaf injury is expected in this field on plants that show symptoms of both leaf spots.

Soybeans as a crop on the Eastern Shore are relatively free of disease at this time. --University of Maryland. July 25, 1946.

AN EPIPHYTOTIC OF ONION MILDEW IN LOUISIANA

E. C. Tims

A destructive epiphytotic of onion mildew (Peronospora destructor) developed in Lafourche and Terrebonne Parishes (two of the most important onion-producing Parishes in the State) this spring. The growers in this area were just getting back into full production of onion again this year after the last disastrous mildew epidemic (1942). The disease caused some damage in most onion-producing sections, but was especially destructive along lower Bayou Lafourche. The unusually warm, wet weather in March was especially favorable for mildew development. More than 15 inches of rain fell in the lower Lafourche section in March. During April, in addition to heavy rains, there were many cloudy, foggy days. The disease spread rapidly and caused most severe damage following heavy rains accompanied by blustery winds.

Mildew was first observed about the first of March in two small fields. By March 15, the disease had spread over widely scattered areas on shallots as well as onions. On March 26, three onion fields were observed with only an occasional mildew-infected plant. The following day a six-inch rain fell accompanied by high winds. A week later (April 2) practically every plant in each of the three fields was diseased. Within two weeks the plants were so badly damaged that practically all growth had ceased.

The numerous small plantings of onions grown for seed were practically wiped out in the Lower Lafourche area. Most of the onions in this area were killed prematurely this year. Mildew caused most of the early killing of the tops, but unusually heavy thrip infestation also caused considerable injury. The losses varied from 40 percent in the lightly infected fields to at least 75 percent in the more severely affected area. The average loss approximated 60 percent in the numerous fields observed in the Terrebonne-Lafourche section with an estimated 1500 acres of onions.

A spray test was carried on this spring in the midst of the worst mildew infected area of the State. A block of onions was selected for the test just across a small ditch from an older onion field that showed heavy mildew infection. When the spray test was started (March 15) there was only an occasional infected plant in the test plots. Sprays were applied at weekly intervals except when heavy rains interfered, until April 23. At this time the plants were so badly injured as to make further applications useless.

Small plots were used consisting of beds 25 feet long with 3 rows of onions on each bed. The plots were randomized and replicated 5 times. The following mixtures were used: Zerlate, 3 lbs. per 100

gal.; COCS spray (3-100); Dithane (2-1 1/2-100); Bordeaux mixture (4-4-50) ready prepared; Lime-sulfur, 2%; Bordeaux 4-4-50 + DDT (2-100); DDT (2-100); and Tennessee copper dust A. A suitable sticker-spreader consisting of rosin soap 1% and a DuPont soap spreader 3/4 lb. to 100 gal. was added to all the spray mixtures. A knapsack sprayer was used and excellent coverage was obtained.

It soon became evident following some heavy rains that none of the sprays was really controlling the mildew, but the test was continued until there was little green color left in any of the plants. The Zerlate, Bordeaux and DDT plots looked somewhat greener than the others all through the test; however, the yields from all the plots were disappointing. Analysis of variance showed no significant difference between the treatments. This test shows that when conditions are especially favorable for mildew development, the disease is very difficult to control.

LOUISIANA AGRICULTURAL EXPERIMENT STATION
BATON ROUGE, LOUSIANA

SOME SHALLOT DISEASES IN LOUISIANA

E. C. Tims

Pink root (Phoma terrestris) is usually the most serious disease of shallot (Allium ascalonicum L.) in the State. However, a pink root-resistant variety of shallot is grown extensively now and the disease is less serious. The Strain 1 variety now known as the "Louisiana Pearl" (3) was developed at the Lousisana Experiment Station some years ago. This variety has a very vigorous root system and although it contracts pink root quite readily, it is not severely injured. The Lousisana Pearl is larger and coarser than the common variety, which has been grown in the State for many years, but in pink root-infected fields it greatly outyields the common shallot.

Virus troubles of shallot are becoming of increasing importance in Lousiana. Shallots are propagated commercially by the use of seed sets and never by true seed. If a lot of shallot sets once becomes infected with a virus it may remain infected for an indefinite period of vegetative propagation. For this reason it is difficult to eliminate viruses except by starting out with seedlings free of disease. Shallots produce seed heads readily, but under Lousiana conditions the seed stalks are very susceptible to injury by various fungi. Considerable difficulty would be encountered in attempting to grow the commercial shallot crop from true seed.

For several years conspicuous, bright yellow bunches have been ob-

served in certain shallot fields (PDR 28: 133, 212. 1944). These yellow bunches may be as tall as the healthy ones, but the leaves are usually small and slender. No mottling or very definite leaf crinkling is associated with this trouble. The amount of infection varied from an occasional plant to as high as 5 percent in one field. Infection has not increased very much during the past several years because the disease is apparently almost self-eliminating. The yellow plants do not form bulbs, and if transplanted to the field they invariably die. Just how the disease is carried from season to season has not been determined, but probably plants infected late in the season carry the virus? through. This yellowing of shallot plants has the appearance of aster yellows. Several bunches of shallots showing the typical yellowing were sent to Dr. Philip Brierley at Beltsville, Maryland, who considered that they were probably infected with aster yellows.

Yellow dwarf (PDR 28: 250, 320, 409. 1944) has become prevalent in many lots of shallot sets and the disease is rapidly becoming a most serious problem in producing this crop. The term yellow dwarf is not quite descriptive of the disease on shallot. There is usually a definite mottling, crinkling and folding downward of the leaves with very little of the bright yellow color associated with the "aster yellows" type of disease. The mottling and crinkling of the leaves lowers the quality of the shallot, but may not make them entirely unfit for market. There is usually a definite reduction in size of the diseased plants, but no comparative yield data have been obtained in Louisiana. According to Brierley (1) a similar disease has been reported in England (2) which causes a marked reduction in yield of shallots. The dry bulbs (sets) from yellow dwarf infected bunches may be normal in size and general appearance.

Several unusual diseases were observed on shallot in Louisiana during the 1945-46 season. The onion purple blotch fungus (Macrosporium [Alternaria] porri) was found causing a conspicuous spotting of shallot leaves in several places. Botrytis sp. caused distinct spotting and discoloration on shallots packed for shipment during the winter. And mildew (Peronospora destructor) was widespread on shallots in some sections of the State.

In November 1945 a representative of the State Department of Agriculture sent in specimens with small, oval, sunken spots 0.5 to 1.5 cm long on the leaves. The color varied from almost perfectly white in the younger spots and around the borders of older spots to a light brown in the center. A few days later similar spots were observed on shallots growing in the Plant Pathology plots at Baton Rouge. The disease was more advanced here, with many conspicuous spots on the shallots in one row. The tips of some leaves were brown and dead. The interior of the spots was a very light brown with a water-soaked border and light yellow area on the outside. The older spots contained numerous spores of the onion purple blotch fungus (Macrosporium [Alter-

naria] porri). The young spots developed spores of the same fungus after two days in a moist chamber. Although this fungus is quite prevalent on onions here, it is seldom found on shallot.

In February 1946, several bunches of shallots were sent in from a packing shed at Plaquemine. The outer scales of some plants were completely discolored, others had light brown spots covered with a prolific development of Botrytis spp. on the surface. This fungus was isolated and compared with a Botrytis from neck rot of onion. The two fungi were quite similar in cultural characters.

The growers said these diseased shallots were grown in seepage spots along the Mississippi River levee. They were probably left in the ground too long. When the demand for shallots falls off and the price drops the plants are often in the field too long and deteriorate to such an extent that they are not suitable for shipping. Often when the price goes back up and the demand increases, the growers go back into the old shallot fields, pick out some of the better bunches and clean them up for market. These old bunches often have a softening of the outer scales. The two most common fungi found growing on such decaying tissues are species of Botrytis and Colletotrichum. The shallots packed for the market with Botrytis lesions had probably been left in the field too long.

Mildew (Peronospora destructor) is not a very common disease of shallot in Louisiana. It does occur to some extent most every year, but during the past several years it has only been observed a few times causing appreciable damage. However, in the spring of 1946, the disease was widespread on shallots in the same general areas where it occurred on onions. Some fields were rather severely damaged, the green leaves being killed back and discolored so badly on many plants as to make them unsuitable for market. Other small shallot plantings were so severely damaged that they were a total loss. Some growers had to pull their shallots prematurely in order to save any of them. The losses from mildew in the shallot-producing areas were not large this year, but the outbreak shows that when conditions are favorable, the shallot may be severely damaged.

There are usually some shallots in the fields from September until about the first of May. Most farmers who raise onions also grow some shallots, usually in the same fields. It seems quite probable that the mildew fungus may be carried through the summer and fall on shallot, either while green in the field or on the dry tops in storage. One of the earliest mildew infections in the Lafourche area this year was found in a shallot field. The disease spread rapidly from the infected shallots to nearby onion fields.

White rot (Sclerotium cepivorum) is confined to a comparatively small area along the east bank of the Mississippi River in St. James

Parish (PDR 26: 219-221. 1942). During the past three years the disease has apparently spread very little, although most of the shallot growers make little effort to prevent it from spreading. If shallots are planted late in the fall in white rot-infested soil so that the plants mature in January or February, the losses are quite heavy, but if the sets are put out in September a good crop may be harvested before conditions are favorable for the disease to develop.

Literature Cited.

1. Brierley, Philip and Floyd F. Smith. Reaction of onion varieties to yellow dwarf virus and to three similar viruses isolated from shallot, garlic and narcissus. Phytopathology 36: 292-297. 1946.
2. Green, D. E. A suspected virus disease of shallots and onions. Jour. Roy. Hort. Soc. 70: 24-29. 1945.
3. Tiebout, G. L. and Joseph Montelaro. Lousiana Shallots. Louisiana State Univ. Ext. Circ. 260. 1946.

LOUISIANA AGRICULTURAL EXPERIMENT STATION
BATON ROUGE, LOUISIANA

OBSERVATIONS ON MISSISSIPPI TOMATO DISEASES DURING 1946

H. H. Foster and J. A. Campbell[1]

For the third consecutive year tomato diseases, aggravated by weather conditions, were important factors contributing to an unfavorable shipping season. Previous reports (PDR 28 (30): 940-944, 1944, and 30 (3): 81-87, 1946) are on record.

The amount and monthly distribution of rainfall for the spring of 1946 is shown in table 1. During January and February, when tomatoes were in the hotbed and coldframe, the excessive precipitation did not seem to affect plant development adversely. Fewer reports were received from growers pertaining to diseases during the seedbed and coldframe period than during the same period in 1945. In general, soil moisture in the Copiah County area was sufficient for field transplanting during the latter part of March and plants became established quickly and initiated new growth in advance of the dry weather that followed. Although the rainfall for April was far below the past average, there was enough moisture in the soil to support good growth. Fruit set well on the first clusters. Rainfall was excessive for May and temperatures were relatively cool during the latter part of the month. An abundance of rain fell during the first ten days of June. During May and June foliage blights and fruit rots became economically important and, directly or indirectly, were factors affecting the quality and value of the commercial tomato crop. The "green wrapped" shipping season opened on May 27, several days earlier than in recent years. Frequently tomatoes were picked prematurely green; otherwise they were of good appearance.

Table 1. Rainfall in inches by months, Spring 1946

	Jan.	Feb.	Mar.	April	May	June	July 1-24
1946	7.36	11.87	4.32	0.20	10.82	2.33	9.76
Normal[a]	5.07	5.27	5.35	5.18	5.34	4.30	5.26

[a] Fifty-year average.

[1] Plant Pathologist and Superintendent, respectively, Truck Crops Branch Experiment Station, Crystal Springs, Mississippi.

Contribution from the Truck Crops Branch Station, Crystal Springs, Mississippi. Published with the approval of the Director, Mississippi Agricultural Experiment Station. Paper No. 121, New Series.

Following are the tomato diseases observed, more or less in order of occurrence:

Collar rot or stem girdling caused by Alternaria solani (Ell. & Mart) Jones & Grout was not observed at the Truck Crops Branch Experiment Station and very few reports regarding this disease were received from tomato growers.

Early blight caused by Alternaria solani was seldom observed during April, and was of no commercial importance. However, as the rainfall and humidity increased during May, early blight infection developed upward through the lower foliage. By June, early blight in connection with other foliage diseases had induced serious defoliation in many commercial fields.

Alternaria fruit spot caused by the early blight fungus was frequently observed. This fruit spot was also prevalent in 1945. Fruit infection appeared near the stem end following injury or fruit cracking, primarily on fruit showing pink. An increase in this fruit spot was observed on the Station plots and at packing sheds late in the season following a foliage blight epidemic caused, in part, by the early blight fungus.

Foliage blight induced by Septoria lycopersici Speg. was not observed in 1946. In both 1944 and 1945 this fungus was abundant in certain plots at the Truck Crops Experiment Station.

Phoma fruit rot caused by Phoma destructiva Plowr. was occasionally observed on pink fruit in experimental field plots and on cull fruit at packing sheds. At the Truck Crops Station tomato foliage occasionally appeared to be infected with the Phoma fungus. Leaves showing rather typical symptoms also showed Phoma spores upon microscopic examination.

Buckeye rot induced by Phytophthora sp., probably Phytophthora parasitica Dast., was again observed on tomato fruit, appearing in certain experimental plots at the Truck Crops Station during the latter half of May. Buckeye was observed primarily on first cluster fruit either touching or near the soil.

During June late blight, Phytopathora infestans (Mont.) DBy., for the most part replaced the buckeye fruit rot. On May 29 the writers inspected two commercial tomato fields west of Crystal Springs, infected with late blight; both foliage and fruit were infected. A few days later late blight was observed at the Truck Crops Station on both foliage and fruit, and by late June it had spread through all experimental plots. Although extensive field surveys were not made, fruits infected with late blight were readily observed at packing sheds during the first two weeks in June, indicating that the epidemic

was of general occurrence throughout the Copiah County area. By the middle of June late blight had reached its peak of development, and most of the foliage had been destroyed by this disease and early blight. Only an occasional fruit infected with late blight was observed during late June; however, reduced but active sporulation was observed on tomato foliage during this period. Higher temperatures together with lower humidity markedly reduced disease development but failed to check sporulation completely.

It is interesting to note that in 1944 late blight was not observed at the Truck Crops Station and reached epidemic proportions only within a limited portion of the commercial area of the State. In 1945 late blight was not reported from the Copiah County area though Buckeye rot was present and of major importance. In 1946 late blight, based on observations and reports reached epidemic proportions as previously mentioned.

In correspondence dated July 1, Dr. G. B. Ramsey, Senior Pathologist, U.S.D.A., reported that in 1946 most of the decay on Mississippi tomatoes arriving on the Chicago market "appeared to be due mostly to late blight and occasionally buckeye rot". He also stated that "the usual secondary bacterial soft rot and Alternaria" were observed "following injuries and bruises especially in the riper fruits".

Several hundred tomato lines and varieties, including selections and crosses, have been under observation at the Truck Crops Station during the past year. All commercial varieties under observation appeared susceptible to both late blight and Alternaria foliage blight. However, a few lines and selections showed some degree of tolerance or resistance to both late blight and Alternaria. Three lines in the Southern Tomato Exchange Program, designated as "STEP" 9, 22, and 23, appeared somewhat resistant to late blight. On June 20, "STEP 9" and "STEP 23" retained approximately the upper one-third of foliage; "STEP 22" appeared superior in resistance. Late blight foliage infection was observed on "STEP 22" but lesions failed to coalesce and no late blight-infected fruits were observed. Earlier, buckeye rot was observed on a number of "STEP 22" fruits. This line (STEP 22) appeared to be segregating for resistance to late blight and Alternaria as certain plants retained foliage better than others. Previously Andrus and Wade (PDR 30 (6): 213-214, 1946) reported "STEP 22" as possessing resistance to late blight.

Certain interspecific crosses (mostly F_2 generation seed) received from Beltsville, Maryland through the courtesy of Dr. W. S. Porte, of the U.S.D.A., produced a few plants that appeared to possess a higher degree of tolerance or resistance to foliage blights (including late blight and Alternaria) than "STEP 22". These plants were vigorous with coarse heavy foliage but with a relatively poor set of small rough fruit.

Plants resistant or tolerant to foliage blights, in all cases, produced a greater number of lateral secondary shoots near the top of the plant giving the general appearance of dense upper foliage. This upper, secondary foliage appeared relatively free from symptoms. Primary foliage on the main stem of "resistant" plants was diseased almost to the same extent as that on susceptible varieties including Rutgers. All plants were pruned to two stems and staked.

The possibility that certain lines are resistant to foliage blights is encouraging. However, at present it is impossible to state that this "bushy" secondary growth of foliage, relatively free from symptoms, represents a sufficiently high degree of resistance to be used satisfactorily in a breeding program.

Fusarium wilt caused by Fusarium bulbigenum var. lycopersici (Brushi) Wr. and Reinking [F. oxysporum f. lycopersici] was observed during the spring season on an occasional Rutgers plant and more generally on highly susceptible varieties such as Bounty, at the Truck Crops Station. No serious reports concerning this disease were received from tomato growers.

The bacterial fruits spots, bacterial spot caused by Xanthomonas vesicatoria (Doidge) Dowson (Bacterium vesicatorium Doidge), and bacterial speck caused by Bacterium punctulans Bryan, were seldom observed in 1946. In 1945 these bacterial diseases were responsible for many cull fruits.

Common mosaic (virus) of tomato was widespread in several of the plots at the Truck Crops Experiment Station and occurred in many commercial fields. Plants infected early in the season were markedly stunted and failed to produce a good set of fruit. This disease is believed to be of greater economic importance than growers realize.

TRUCK CROPS BRANCH EXPERIMENT STATION
CRYSTAL SPRINGS, MISSISSIPPI

TOMATO LATE BLIGHT ON THE VIRGINIA EASTERN SHORE

OBSERVATIONS ON INCIDENCE OF LATE BLIGHT IN MID-JULY

By L. P. McColloch

In the Plant Disease Reporter Supplement 164, issued July 15, 1946 Dr. Harold T. Cook of the Virginia Truck Experiment Station reported on late blight (Phytophthora infestans) of potatoes and tomatoes in Eastern Virginia. The following observations were made on the Eastern Shore of Virginia, July 16 to 18, while attending a Federal-State Inspectors conference, and are offered as a supplement to those published by Dr. Cook. Credit is due to M. E. Downing, agent for the Federal-State Inspection Office at Cape Charles, Virginia, and to H. Marshall Clark, County Agent of Accomac County, for much of this information.

It was understood that late blight on tomatoes was serious throughout Accomac County, but that in Northampton County the outbreak was worse in the extreme northern and southern portions than in the county as a whole.

Hot, dry weather just prior to July 15 had arrested the disease and lesions on leaves were dry and brittle. All stages of the disease were found on the fruits. There was a great deal of variation in its occurrence in the fields, however. In some it was difficult to find a sound tomato, while in other fields diseased fruits were rarely found. Estimates on losses vary from 1/3 to 1/2 of the normal yield.

A few fields were seen that had been dusted by means of an aeroplane. Very little late blight was found in these, but it would be difficult to evaluate the effect of the fungicide because the disease was arrested by weather conditions very soon after the application of dust.

There was considerable discussion as to the cause of such an extensive outbreak of late blight and the source of infection. It was the consensus that the seriousness of the disease was due to cool rainy weather. Regarding the source of infection, most people interviewed thought that the late blight had spread from the potatoes to the tomatoes. One can only speculate at this time about the source of infection on the potato crop, but I am inclined to think that it is carried over in certain fields. The first outbreak of late blight occurred in the southernmost part of Northampton County, which is separated from vegetable crops to the south and the mainland of Virginia by a distance of 30 miles. It does not seem likely that viable spores of this disease would be carried by the wind that distance across the Capes. An extensive acreage of potatoes is grown in this area, and the crop is quite often planted year after year on the same soil. A mild in-

fection of late blight in the potato crop is not unusual. Tomatoes are frequently planted following potatoes and this year numerous volunteer potatoes were observed in tomato fields. Such cultural practices seem sufficient to keep a mild infection present, but an epidemic would depend upon cool weather and excessive rainfall. -- Division of Fruit and Vegetable Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Beltsville, Maryland.

DECREASE IN TOMATO LATE BLIGHT
IN THE LATTER PART OF JULY

By Harold T. Cook

A survey of the tomato crop on the Eastern Shore of Virginia, July 25, showed that late blight had nearly disappeared in the fields on the lower half of the Peninsula and that it was spreading more slowly in the northern part. Excellent quality tomatoes were being harvested.

Loss from late blight in the early green wrap crop probably amounted to 75 percent. Very little loss is now expected in the later plantings.

Higher temperatures and less rain are credited with stopping the spread of late blight. -- Virginia Truck Experiment Station, Norfolk. July 31

OTHER REPORTS OF LATE BLIGHT ON POTATO AND TOMATO

LATE BLIGHT ON TOMATO
IN WAYNE COUNTY, NEW YORK

By Leo Dillon

Late blight is present in very serious proportions, already having ruined some fields beyond possible salvage. To show how rapidly the disease can spread under favorable conditions, in one field which on the morning of August 5 showed only leaf and slight stem infection, more than 90 percent of the fruit was infected by the night of August 7, in spite of the fact that the field had been sprayed in the meantime. -- From New York State College of Agriculture Weekly News Letter on Insect Pests and Plant Diseases, August 12

TOMATO LATE BLIGHT IN MARYLAND

By R. A. Jehle and F. C. Stark, Jr.

Severe losses from late blight are being sustained by tomato growers in Maryland. On July 26 it was estimated that in the southernmost Eastern Shore counties, Worcester, Somerset, and Wicomico, 85 percent of the crop would be lost due to infection of the fruit. In portions of the State bordering Pennsylvania losses from fruit infection are estimated at 20 to 25 percent. In other parts of the State losses are estimated at 10 to 20 percent of the crop. If weather conditions are normal, they will be favorable for further development of late blight and other rots, and greater losses may be expected. Losses from late blight in tomatoes are usually negligible in Maryland and this is the most severe loss from this disease sustained by tomato growers in the past 25 years. -- University of Maryland, July 27.

POTATO LATE BLIGHT IN WISCONSIN FIELDS

By R. E. Vaughan

Potato late blight was observed in the field in Langlade County, Wisconsin, July 10, following several days of rainy weather. It was showing slightly in several fields July 16 to 19. Commercial growers are doing a good job of spraying with Bordeaux mixture or dusting with the tribasic copper compounds on the market. Many are using DDT in the early applications to control leafhopper. Dithane spray is being tried by a number of growers. -- University of Wisconsin, July 23

LATE BLIGHT IN PENNSYLVANIA: A CORRECTION

The date July 8 cited for appearance of late blight on potato in Pennsylvania in the report of R. S. Kirby, in Supplement 164, page 289, is an obvious typographical error. The correct date is June 8.

DOWNY MILDEW ON CUCURBITS

RAPID SPREAD OF DOWNY MILDEW ON CUCUMBER AND CANTALOUP ON THE MARYLAND EASTERN SHORE

By C. E. Cox

Downy mildew (Pseudoperonospora cubensis) is taking a heavy toll of cucumbers and cantaloups on the Eastern Shore of Maryland. During the

heavy rains of the week of July 22 the disease has spread to new foliage and an epiphytotic is inevitable. Counties suffering worse from this disease are Dorchester, Wicomico, Worcester, Talbot, and Caroline, according to field observations of E. A. Walker and W. F. Jeffers.

Cucumbers are more severely affected. In most fields observed in the area leaves are heavily spotted, and the fields are taking on a yellow appearance as the foliage responds to the effect of the fungus. The first picking of cucumbers is completed in most affected areas. Some growers will make a second picking, after which the crop will be lost by defoliation, sunscorch, and "nubbin" fruit.

Downy mildew on cantaloups has not developed so far, and there is prospect of controlling further development if growers will apply a copper fungicide (5 to 7% metallic copper) to their fields at once. On cantaloups, downy mildew has killed some center leaves and has spread onto the main crop leaves as new infection centers. Harvest of cantaloups has just begun on the Eastern Shore and the early picking will not be affected. If the disease is not controlled, however, it may destroy the crop before the main harvest season arrives.

Downy mildew on cantaloup leaves produces round spots with a brown center surrounded with a lighter yellow halo, while on cucumber leaves the spots are round with yellow centers with indefinite margin. -- University of Maryland, July 24

CUCUMBER DOWNY MILDEW IN MASSACHUSETTS

By O. C. Boyd

Following a recent report of the finding of cucurbit downy mildew in Massachusetts, I visited fields in Bristol and Plymouth Counties this week, the parts of the State where the disease usually appears first. On August 7, all older cucumber plantings (where harvesting had been under way for one to two weeks) were moderately to heavily infected, usually showing two or three early-infected areas where the plants were entirely dead, with secondary infection over the rest of the field. The younger plantings, just coming into picking or not that far along yet, showed mostly light infection that appeared to be secondary infections from nearby older fields. Two or three such plantings showed no downy mildew yet. The only older fields that were not infected had been rather well dusted or sprayed with copper fungicides.

The disease was first observed in Bristol County by County Agent Chas. Harris, Jr., on July 30, and one week earlier by the grower where Harris collected the specimens for sending to me. On one farm, where overhead irrigation had been practiced, the disease was uniformly

severe over the field and was also present on an adjacent planting of cantaloups.

On July 8, several cucumber fields in Plymouth County were visited. Here, the growers are much more concerned with spraying or dusting their cucumbers. Most of the unprotected older fields showed recent infections, probably coming from older infections in Bristol County, but most of the plantings in Plymouth County had been well dusted or sprayed with copper and showed either no disease or very light infections. Only one case was observed where the original infection was probably as early as the numerous early cases in Bristol County, and that one case had not spread out into the field very far since the vines had been dusted regularly.

One grower in Bristol County is using a 10% Fermate-talc dust on a young planting of cucumbers, starting ahead of the disease.

No downy mildew has been seen yet in the Connecticut Valley. -- Massachusetts State College, August 10

SOME BACTERIAL DISEASES OF TRUCK CROPS IN EASTERN VIRGINIA

Harold T. Cook

CUCUMBER Approximately 10 percent of the cucumbers in the vicinity of Bowling Green in Caroline County were affected with bacterial wilt (Bacillus tracheiphilus) [Erwinia tracheiphila] on July 16

PEPPER Peppers in Caroline County were found to be badly damaged by bacterial leaf spot (Bacterium vesicatorium) [Xanthomonas vesicatoria] on July 16. It is understood that about 200 acres of this crop was contracted with two canning companies and that the seed was furnished by those companies. -- Virginia Truck Experiment Station, Norfolk. July 18

CHECK LIST REVISION

Freeman Weiss

CHENOPODIUM (CHENOPODIACEAE)

CHENOPODIUM ALBUM L., GOOSEFOOT, LAMB'S-QUARTERS. Cosmopolitan weed, annual; sometimes collected for greens.

Albugo bliti (Biv.-Bern.) Kuntze, white rust. Miss., Tex. Reports of A. candida on Chenopodium probably belong here, but may refer to Peronospora.

Cercospora beticola Sacc., leaf spot. Iowa. Natural infection observed and susceptibility of this host as well as beet demonstrated.

C. dubia (Riess.) Wint. (C. chenopodii Fres.), leaf spot. General from Mass. to S. Car., Mo. and N. Dak.

Diplodia natalensis Pole-Evans (? Physalospora rhodina (Berk. & Curt.) Cke.), on stems. Ala.

Diplodina ellisii Sacc. (D. hyalospora Cke. & Ell.), on stems. N. Y.

Ditylenchus dipsaci (Kuehn) Filip., stem nematode. N. Y.

Dothidella longissima (Pers. ex Fr.) Ell. & Ev., on stems. Kans.

Gloeosporium melleum Dearn. & Overh., leaf spot. Pa.

Hadrotrichum chenopodinum Pk., on leaves. N. Y.

Heterodera marioni (Cornu) Goodey, root knot. Okla., S. Car., Wis.

Leptostromella chenopodii Dearn. & House, on stems. N. Y.

Metasphaeria anthelmintica (Cke.) Dearn., on stems. N. Y.

Peronospora sp. (spp.), downy mildew. Widespread, chiefly in the North, from Mass. to Va., Kans., and Wash.; occasional in the South -- Miss., Okla., Tex. Two spp. with a common or overlapping range have been reported but not always distinguished: P. effusa (Grev.) Rab. and P. farinosa Fr. (P. chenopodii Schlecht.). The former, also called P. variabilis Gäum., is at least physiologically different from P. effusa on spinach.

(Phyllosticta chenopodii West.): Stagonospora atriplicis.

P. chenopodiicola Tehon & Daniels reported in Ill. and P. chenopodii-albae Siem. in Wis. may belong here

Phymatotrichum omnivorm (Shear) Dug., root rot. Tex.

Pratylenchus pratensis (De Man) Filip., root nematode. Utah

Puccinia aristidae Tracy (O,I), rust. Kans. and N. Dak. to Ariz. and Mont., also Ind. II and III on many grasses.

Rhizoctonia solani Kuehn, root rot. Wash.

Sclerotinia sclerotiorum (Lib.) DBy., root rot. Mont.

Stagonospora atriplicis (West.) Lind, leaf spot. General. Variously reported as Ascochyta, Phyllosticta, Phleospora, Septogloeum, and Septoria.

Chenopodium album cont.

Uromyces peckianus Farl. (0,I), rust. Calif. II and III on various grasses. Correlated with Puccinia aristidae

CHENOPODIUM SPP. Indigenous or adventive annuals, as C. CAPITATUM (L.) Asch. (1), C. GLAUCUM L. (2), C. HYBRIDUM L. (3), C. LEPTOPHYLLUM Nutt. ex S.Wats (4), C. RUBRUM L. (5). Of some forage value and furnishing food for wildlife; sometimes used for greens. C. AMBROSIOIDES L., especially the var. ANTHELMINTICUM (L.) A.Gray, AMERICAN WORMSEED, MEXICAN-TEA (6), a perennial of tropical America, is cult. as a medicinal plant.

Albugo occidentalis G.W.Wils., white rust. Colo., Iowa, Wyo. (1), Mont. (5)

Cercospora anthelmintica Atk., leaf spot. N. J. to Ala. and Tex. (6).

C. dubia (Riess) Wint., leaf spot. Iowa, Kans., Okla. (3); Kans., Nebr. (4), N. Dak. (5), Tex. (sp.), Wis. (1,3)

Diaporthe arctii (Lasch) Nits., on stems. Ga. (6)

Fusarium pallidoroseum (Cke.) Sacc., on stems. Md. (6)

Hendersonia bliti Clements, leaf spot. Colo. (1)

Heterodera marioni (Cornu) Goodey, root knot. Ala. (6)

H. schachtii A.Schm., root gall. Utah (5)

Leptosphaeria anthelmintica (Cke.) Sacc. N.Dak. (2)

Leptothyrium chenopodii Dearn. & Barth., on stems. Kans. (3)

Mycosphaerella chenopodii Dearn. & Barth., on stems. Kans. (4)

Peronospora effusa (Grev.) Rab., downy mildew. Ill., Iowa, Kans., Nebr., S. Dak., Wis. (3). P. chenopodii-glauci Gäum. on (2) and (4) in Kans. has also been reported.

P. farinosa (Fr.) Keissler (P. chenopodii Schlecht.), downy mildew. Iowa (3)

Phyllosticta ambrosioides Thuem. & Syd., leaf spot. Del., Ill., Ind., La., N. J., N. Y. (6)

Puccinia aristidae Tracy (0,I), rust. Ariz. (2,4,5); Calif. (sp.), Colo. (1,2,3,4,5), Mont. (2), Nebr. (3,4), N. Mex. (2,4,5), Utah (4), Wyo. (5).

Pyrenophora calvescens (Fr.) Sacc., on stems. N. J., N. Y. (spp); W. Va. (6)

Rhizoctonia solani Kuehn, damping-off. Kans. (4)

Stagonospora striplicis (West.) Lind, leaf spot. Mich., Minn., Wis. (1); N. Dak. (5); N. Y. (on C. bonus-henricus L.)

Uromyces peckianus Farl. (0,I), rust. On C. murale L.

Urophlyctis pulposa (Wallr.) Schroet., leaf and stem gall. S. Dak. (2)

Curly top -- virus (Chlorogenus eutetticola Holmes, Beta virus 1 K.M.Sm.). Natural infection of (4), (6), and C. murale reported in Calif.

KOCHIA (CHENOPODIACEAE)

KOCHIA SCOPARIA (L.) Schrad., SUMMER-CYPRESS. Old World annual, cult. for ornament, especially the var. TRICHOPHYLLA Bailey; sometimes escaped and locally established.

Phymatotrichum omnivorum (Shear) Dug., root rot. Tex.
Puccinia aristidae Tracy (O,I), rust. Colo. Nebr.

Curly top -- virus (Chlorogenus eutetticola Holmes, Beta virus 1 K.M.Sm.). Calif.

SALICORNIA (CHENOPODIACEAE)

SALICORNIA SPP., GLASSWORT. Chiefly S. EUROPEA L., an annual herb, and S.PERENNIS Mill., a woody perennial, widely distributed salt-marsh plants, of food value to wildlife.

Mycosphaerella salicorniae (Auers.) Lindau, on stems. Conn.
Pleospora salsolae Fckl., on stems, Mass.
Protomyces conglomeratus Pk., stem gall. N. Y.
Uromyces peckianus Farl. (O,I), rust. Calif., Wash. II and III on Aristida and Distichlis

SALSOLA (CHENOPODIACEAE)

SALSOLA KALI L., SALTWORT, especially the var. TENUIFOLIA Tausch (S. PESTIFER A. Nels.), RUSSIAN-THISTLE. Cosmopolitan annual of saline or alkaline soils, often a troublesome weed on range lands, but of some forage value when young.

Melanospora tomei Griff., on stems. S. Dak.
Phymatotrichum omnivorum (Shear) Dug., root rot. Ariz.
Pleospora lecanora (Fabre) Rehm, on stems. N. Dak.
Puccinia aristidae Tracy (O, I), rust. Ariz., Colo., Nebr., N. Mex., Utah.
Pyrenophora salsolae Griff., on stems. N. Dak.

Curly top -- virus (Chlorogenus eutetticola Holmes). Calif.

SPINACIA (CHENOPODIACEAE)

SPINACIA OLERACEA L., SPINACH. Annual, of S.W. Asia, widely cult. for greens; an important truck and canning crop in Tex., Cal., Okla., Va., N. Y., N. J.

Spinacia oleracea cont.

Albugo occidentalis G.W.Wils., white rust. Ark., Okla., Tex., ? Va.

Alternaria sp. (probably A. tenuis Nees ex Wallr.), seed discoloration, secondary leaf spot. Conn., Ohio, S. Car.

Cercospora beticola Sacc., leaf spot. Ga., Ill., Iowa, Mass., N. Y., Tex. Perhaps distinct, though probably at times confused with this sp., is C. flagilliformis Ell. & Halst., a nomen nudum, reported in Del., Ga., Ind., Minn., and N. J. C. beticola from Beta vulgaris has been shown pathogenic to spinach. C. dubia (Riess) Wint. is also reported on this host in Mo.

Cladosporium macrocarpum Preuss, leaf mold (secondary). Del., Okla., Pa., Tex.

C. subnodosum Cke. Del., S. Car.

Colletotrichum spinaciae Ell. & Halst., anthracnose. Conn., La., Miss., N. J., N. Y., Tex., Va.

Entyloma ellisii Halst., leaf smut. N. J.

Erwinia carotovora (L.R.Jones) Holland, bacterial soft rot. Occasional in transit and markets.

Fusarium spp., root rot, crown rot, wilt. General. A form of F. solani (Mart.) App. & Wr. has been particularly cited as associated with spinach wilt in Tex.; F. oxysporum Schlecht. f. spinaciae (Sherb.) Snyder & Hansen is reported from Calif., Conn., Ida., and Tex.

Heterodera marioni (Cornu) Goodey, root knot. Ga., Ind., S. Car., Va.

Heterosporium variabile Cke., leaf spot (pinhead "rust"). Widespread.

Peronospora effusa (Grev. ex Desm.) Ces. in Rab., (usually cited (Grev.) Rab.), downy mildew. General. The form on spinach is said to differ morphologically and physiologically from that on Chenopodium and other genera of the family, and is therefore entitled to specific rank as P. spinaciae Laub. P. effusa was originally described on spinach and, though not recognized by Fries, the name was satisfactorily validated by Desmazières. If any different name is required the form on Chenopodium should bear it, that on spinach remaining P. effusa. This need is perhaps adequately met by P. farinosa Fr., which is sometimes but unnecessarily cited (Fr.) Keissler.

Phoma terrestris Hansen, secondary root rot. Iowa

Phyllosticta chenopodii Sacc., leaf spot. Del., N. J., N. Y., Va.

Phymatotrichum omnivorum (Shear) Dug., root rot. Tex.

Phytophthora sp., crown rot. Ind., N. J., N. Y.

P. drechsleri Tucker, root rot. Hawaii.

P. megasperma Drechs., root rot. Calif., N. Car.

Pleospora herbarum (Pers. ex Fr.) Rab. (Stemphylium botryosum Wallr.), seed discoloration, mold.

Puccinia aristidae Tracy (0,I), rust. Ariz., Calif., Oreg., Wash. II and III on grasses, commonly Distichlis.

Spinacia oleracea cont.

Pythium spp., seed decay, pre-emergence damping-off, root rot. Calif., Northeastern and Central States. Commonly reported as P. debaryanum Hesse. P. anandrum Drechs., P. aphanidermatum (Edson) Fitz., P. helicoides Drechs., P. irregulare Buis., P. polytylum Drechs., P. rostratum Butl., and P. ultimum Trow also have been identified in Calif.; P. dissotocum Drechs. in Va.; P. vexans DBy. in Hawaii.

Rhizoctonia sp. (R. solani Kuehn), damping-off, root rot. General

Sclerotinia sclerotiorum (Lib.) DBy, crown rot. Ga., N. Y.

Stagonospora spinaciae Ell. & Ev., leaf spot. S. Dak.; also ? Tex. as Septoria or Phyllosticta sp.

Verticillium sp., wilt. N. Y.

Curly top -- virus (Chlorogenus eutetticola Holmes, Beta virus 1 K.M.Sm.). Calif., Oreg., Tex., Utah, Wash.

Mosaic (blight) -- virus (in part Marmor cucumeris Holmes, Cucumis virus 1 K.M.Sm.). Reported especially in the Middle Atlantic and Southern States, occasionally from all other spinach-growing areas except the Pacific Northwest. Besides the cucumber mosaic virus, the viruses of beet mosaic, turnip (cabbage) mosaic, and tobacco ringspot can induce systemic infection in spinach; one or more of these may be involved in field infections of mosaic.

Spotted wilt -- virus (Lethum australiense Holmes, Lycopersicon virus 3 K.M.Sm.). Calif., Hawaii

Yellows -- in part, virus (Chlorogenus callistephi Holmes, Callistephus virus 1 K.M.Sm.). N. J., Tex. In part physiological, attributed to nutrient deficiency, as of magnesium (N. J., N. Y., Va.), manganese (N. J., N. Y., R. I.), or undetermined (Conn., Mass., Md., Ky., S. Car., Tex., Va., Wash.)

ARGYREIA (CONVOLVULACEAE)

ARGYREIA NERVOSA Bojer (1) and A. SPECIOSA Bojer (2). Asiatic twining perennials, grown for ornament.

Heterodera marioni (Cornu) Goodey, root knot. ? S. Car. (1).

Phymatotrichum omnivorum (Shear) Dug., root rot. Tex. (2)

CALONYCTION (CONVOLVULACEAE)

CALONYCTION ACULEATUM (L.) House (Ipomoea bona-nox L.), MOONFLOWER. Twining perennial of tropical America, grown in warm regions or under glass for ornament.

Albugo ipomoeae-panduratae (Schw.) Swing., white rust. Fla.

Calonyction cont.

Aphelenchoides fragariae (Ritz.-Bos) Christie (group), leaf nematode. N. J.
Coleosporium ipomoeae (Schw.) Burr. (II, III), rust. Ala., N. and S. Car., Tex. O and I on Pinus spp.
Heterodera marioni (Cornu) Goodey, root knot. N. J., ? S. Car.
Phyllosticta sp., leaf spot. N. J.
Phymatotrichum omnivorum (Shear) Dug., root rot. Tex.
Ramularia ipomoeae F.L.Stevens, leaf spot. Hawaii
Sphaerulina ipomoeae F.L.Stevens, on leaves. Hawaii

CONVOLVULUS (CONVOLVULACEAE)

CONVOLVULUS ARVENSIS L., SMALL BINDWEED (1), C. SEPIUM L., GREAT or HEDGE BINDWEED (2). Twining perennials, (1) naturalized from Europe throughout the U. S., a persistent weed; (2) indigenous in the Eastern and Central States, of some food value to wildlife and sometimes used for ground cover. Also several native (e.g. C. californicus Choisy, C. occidentalis Gray (3)) and introduced (C. japonicus Thunb. (4)) spp., sometimes grown for ornament.

Albugo ipomoae-panduratae (Schw.) Swing., white rust. N. J. to Fla., Tex. and Ill. (2); Tex., P. R. (3)
Cercospora sp., leaf spot. N. Car. (2)
C. tuberculella J.J.Davis. Wis. (2)
Coleosporium ipomoeae (Schw.) Burr. (II,III), rust. N. Car. (1); Ala., Ga., Md., Tenn., Va., W. Va. (2); La., Miss., (3). O and I on Pinus spp.
Cuscuta gronovii Willd., dodder. N. Y. (2)
Diplodia convolvuli Dearn. & House, on stems. N. Y. (2)
Dothidella calystegiae (Cke. & Harkn.) Theiss. & Syd., on stems. Calif. (3)
Heterodera marioni (Cornu) Goodey, root knot. Md. (4)
Mycosphaerella sp. (Sphaerella harknessii Saec.), on stems. Calif. (3)
Parodiella paraguayensis Speg., black mildew. N. Car. (1)
Phyllosticta batatas (Thuem.) Cke., leaf spot. S. Car. (3)
Phymatotrichum omnivorum (Shear) Dug., root rot. Tex. (1,2,3).
Physalospora obtusa (Schw.) Cke., on stems. (2)
Puccinia convolvuli (Pers.) Cast. (0,I,II,III), rust. Calif., Ind., Mass., Nebr., N. Y., Okla., Tenn. (1); Mass. to Ala., Kans., and N. Dak. (2); Calif., Oreg., Virgin Is. (3)
Rhabdospora sp., stem rot, blight. Ida., Wash. (1)
Rhizoctonia solani Kuehn, damping off. Minn. (1)
Septogloeum convolvuli Ell. & Ev., leaf spot. Calif., Wis. (3)
Septoria calystegiae West., leaf spot. Wis. (1), N. Y. (2), Calif. (3)

Convolvulus cont.

Septoria convolvuli Desm. Vt. to Md., Colo. and Wash. (2); Wis. (3). Reported also on (1) but probably confused with S. septulata.

S. flagellaris Ell. & Ev. Mich., Miss., N. J., N. Y. (2); N. Y. (3)

S. septulata Beach. Ill., Nev. (1); probably also Iowa, Kans., Nebr., N. J., Wis. reported as S. convolvuli; Wash. (3)

Stagonospora convolvuli Dearn. & House, leaf spot. N. Y. (2)

DICHONDRA (CONVOLVULACEAE)

DICHONDRA CAROLINENSIS Michx., LAWN--LEAF (1) and D. REPENS Forst. (2). Creeping perennials sometimes used for ground cover and lawns in the far South.

Heterodera marioni (Cornu) Goodey, root knot. Calif. (2)

Puccinia dichondrae Mont. (I,III), rust. La., Miss., N. Car., Tex. (1); Calif., Tex. (2)

Sclerotium rolfsii Sacc., southern blight. Calif. (2)

Synchytrium edgertonii Cook, leaf gall. La. (2)

EVOLVULUS (CONVOLVULACEAE)

EVOLVULUS SPP. Low perennials, mostly in the Western Plains, of food value to wildlife.

Puccinia lithospermi Ell. & Kell. (0,I,II,III), rust. Colo., Kans., Nebr., Tex., P. R.

IPOMOEA (CONVOLVULACEAE)

IPOMOEA BATATAS (L.) Lam., SWEETPOTATO. Tropical American perennial, widely cult. for edible roots; in the U. S. grown in the Southern States north to N. J. and Iowa, also Ariz., Calif., Wash., Hawaii, and P. R.; commercially chiefly from N. J. to Fla., Tex. and Mo.

Actinomyces ipomoea Person & W.J.Martin, soil rot (pox, pit). General from N. J. to Fla., Tex., and Iowa, also Ariz. and Calif. The stated distribution refers to the disease, not to this specific organism which has been identified only in La., but it is believed to be identical with Actinomyces pox Manns & Adams (a nomen nudum) and Actinomyces p. Adams. Other Actinomyces spp. as A. poolensis Taub. and A. scabies (Thaxt.) Güssow, sometimes associated with soil-rot lesions, are secondary. A Streptomyces in the sense of Waksman & Henrici but

Ipomoea batatas cont.

not yet so combined.

Albugo ipomoeae-panduratae (Schw.) Swing., white rust. General

Alternaria sp. (? A. tenuis Nees ex Wallr.), secondary leaf spot. Occasional to general, often following white rust. Storage rot of roots, also attributed to Alternaria, may be caused by other spp. A. solani (Ell. & Mart.) Jones & Grout is reported to cause foot rot in Del. and Tex.

Aspergillus spp., especially A. niger v. Tiegh., secondary storage rot. Occasional.

Botrytis cinerea Pers. ex Fr., gray mold rot. On sprouts in hotbeds and in stored roots, usually after chilling. Cosmopolitan

(Ceratostomella fimbriata (Ell. & Hals.) J.A.Elliott): Endoconidiophora f.

Cercospora sp., leaf spot. Fla., Okla., P. R. Identified doubtfully as C. batatae Zimm. (which occurs in Guam and the Philippine Is.); also reported as C. ipomoeae Wint. and C. bataticola Cif. & Bruner

Choanephora cucurbitarum (Berk. & Rav.) Thaxt., leaf mold. Fla.

Colletotrichum sp., leaf spot. Miss.

Coleosporium ipomoeae (Schw.) Burr. (II,III), rust. Ala., Miss., P. R.

Diaporthe batatatis Harter & Field, dry rot (roots), stem rot. N. J. to Fla., Tex. and Mo., especially in the South. (D. phaseolorum (Cke. & Ell.) Sacc. var. batatatis (Harter & Field) Wehm.)

Diplodia tubericola (Ell. & Ev.) Taub. (= D. theobromae (Pat.) Nowell), Java black rot (of roots). General, especially in the South. Conidial stage of Physalospora rhodina (Berk. & Curt.) Cke.

Ditylenchus dipsaci (Kuehn) Filip., brown ring (roots). N. J.

Endoconidiophora fimbriata (Ell. & Hals.) Davidson, black rot (roots and stems). General

Epicoccum sp., storage rot. Occasional after chilling.

Erwinia carotovora (L.R.Jones) Holland, bacterial soft rot. Conn., S. Car.

Fuligo violacea Pers., epiphytic on sprouts in hotbeds. Ala., Del., N. J.,Tex.

Fusarium oxysporum Schlecht., surface rot (of roots in storage). General.

F. oxysporum f. batatas (Wr.) Snyder & Hansen (F. batatatis Wr., F. hyperoxysporum Wr.), stem rot, wilt. General, especially in the northern range of sweetpotato production, including Pacific Coast and Western States; in the South on susceptible varieties. The organisms originally distinguished as F. batatatis and F. hyperoxysporum (subsequently as F. bulbigenum Cke. & Mass. var. batatas Wr. and F. oxysporum Schlecht. f. 2 Wr.) may be regarded as physiological races of F. oxysporum, differing from races that cause surface rot.

Ipomoea batatas cont.

Fusarium spp. The following spp. have also been reported, causing secondary storage rot (end rot and some forms of dry rot): F. culmorum (W.G.Sm.) Sacc., F. scirpi C.R.Lambert & Fautr. var. acuminatum (Ell. & Ev.) Wr., and F. semitectum Berk. & Rav. var. majus Wr. -- all synonyms of F. roseum Lk. emend. Snyder & Hansen and conidial stages of Gibberella saubinetii (Mont.) Sacc.; F. solani (Mart.) App. & Wr. and F. javanicum Koord. var. radicicola Wr. -- conidial stages of Hypomyces ipomoeae (Hals.) Wr.

Helicobasidium purpureum (Pat.) Tul., root rot. Kans., Tex.

Heterodera marioni (Cornu) Goodey, root knot. General from N. J. to Fla., Tex. and Mo.; Calif. Porto Rico and Big-Stem Jersey types resistant.

Hypomyces ipomoeae (Hals.) Wr., on stems and roots following wilt infection and storage rot. Calif., Del., Md., N. J., Ohio, S. Car. See Fusarium solani

Macrophomina phaseoli (Maub.) Ashby, charcoal rot (of stored roots), stem rot. N. J. to Fla., Tex., and Kans.; Calif.

Meliola clavulata Wint., and M. malacotricha Speg., black mildew. P. R.

Monilochaetes infuscans Ell. & Hals. ex Harter, scurf. General

Mucor racemosus Fres., storage rot. Occasional after chilling. Cosmopolitan

(Nectria ipomoeae Hals.): Hypomyces i.

Penicillium sp., blue mold rot. On stored roots, after chilling or wounding. Cosmopolitan.

Pestalotia batatae Ell. & Ev., on roots. Ala.

Phoma terrestris Hansen, secondary (?) root spot. Calif.

(Phomopsis batatae (Ell. & Hals.) Harter & Field): Diaporthe batatatis

Phyllosticta batatas (Thuem.) Cke., leaf blight. Occasional, N. J. to Fla., Tex. and Kans., usually in the South.

Phymatotrichum omnivorum (Shear) Dug., Texas root rot. Ariz., N. Mex., Okla., Tex.

Physarum spp. (P. cinereum (Pers.) Batsch, P. plumbeum Fr.), epiphytic on sprouts in hotbeds. Ala., Ark., Ky., Miss., N. J., Va.

Phytophthora sp., root rot. N. J., Va.

Plenodomus destruens Harter, foot rot (of stems and roots), sometimes storage rot. N. J. to Fla., La. and Kans.; Calif.

Pythium spp., rootlet rot, mottle necrosis of mature roots, leak and ring rot in transit and storage. P. scleroteichum Drechs. and P. ultimum Trow, perhaps sometimes P. debaryanum Hesse, are the principal spp. causing mottle necrosis, reported in Calif., Del., Ind., Iowa, Md., Miss., N. J., N. Car., Pa., Va., Wash.; possibly also P. aphanidermatum (Edson) Fitz. and P. splendens Braun. P. ultimum especially and sometimes other spp. cause rootlet rot, widespread in seedbeds, sometimes in fields. P. aphanidermatum and P. ultimum are reported causing

Ipomoea batatas cont.

leak in Calif., the latter also ring rot. P. arrhenomanes Drechs., P. artotrogus (Mont.) DBy., P. irregulare Buis., P. rostratum Butl., associated with rootlet rot in Hawaii.

(Rhizoctonia crocorum DC. ex Fr.): Helicobasidium purpureum

R. solani Kuehn, sprout rot, rootlet rot, stem canker. Occasional in different forms, N. J. to Fla. and Tex.; Ariz., Calif., Ohio, Wash.

Rhizopus spp., soft rot, ring rot (of stored roots). General. R. stolonifer (Ehr. ex Fr.) Lind (R. nigricans Ehr.) is the most common cause of soft rot at medium to low temperatures, R. tritici K. Saito at higher.

Rotylenchus similis (Cobb) Filip., root nematode. Hawaii

Schizophyllum commune Fr., dry rot of roots. N. Car., P. R.

Sclerotinia sp., storage rot following chilling. N. Car.

S. minor Jagger, sprout rot in seedbeds. N. J.

(Sclerotium bataticola Taub.): Macrophomina phaseoli

S. rolfsii Sacc., southern blight, cottony rot (commonly in seedbeds, sometimes in fields). General in the South, Va. to Fla., Tex. and Okla.

Septoria bataticola Taub., leaf spot. Occasional, N. J. to Ala., Tex. and Iowa; Hawaii

Sphaceloma batatas Saw., scab, bud stunt. Guam

(Sphaeronema fimbriatum (Ell. & Hals.) Sacc.): Endoconidiophora fimbriata

Trichoderma spp. (mostly T. viride Pers. ex Fr.), wound rot of stored roots. Del., N. J., N. Car.

Verticillium albo-atrum Reinke & Berth., wilt. Calif.

Curly top -- virus (Chlorogenus eutetticola Holmes, Beta virus 1 K.M.Sm.). Oreg., Tex.

Fasciation (flat and ring types) -- physiological, perhaps due to over-nutrition. Occasional everywhere.

Feathery mottle -- virus (Flavimacula ipomoeae Doolittle & Harter). Md. (in experimental plants).

Internal breakdown (of stored roots) -- physiological, perhaps resulting from prolonged storage at high temperature and low humidity. Occasional.

Internal cork -- suspected virus. Miss., S. Car.

Internal brown spot -- boron deficiency. In experimental plots. S. Car.

Intumescence (leaf overgrowths, ? raised veins) -- physiological, attributed to high water intake and retarded transpiration. Occasional

Mosaic -- suspected but unconfirmed virus. Occasional on Nancy Hall var. Va. to Fla., Tex., and Kans.; Iowa, N. Mex. Infection with a virus believed to be a strain of cucumber mosaic virus (Marmor brassicae Holmes) has been reported in Fla. Variegation of genetic origin occurs occasionally.

IPOMOEA HEDERACEA (L.) Jacq., IVYLEAF MORNING-GLORY (1); I. NIL (L.) Roth, WHITE-EDGE M. (2); I. PURPUREA (L.) Lam., COMMON M. (3); I. TRICOLOR Cav. (4); other spp. (5). In improved forms, the annual morning-glories of floriculture, natives of tropical America; (1) widely and (3) locally naturalized in the U. S., the former especially becoming a weed.

Albugo ipomoeae-panduratae (Schw.) Swing., white rust. N. J. to Ala., Ariz., and Nebr. (1,2,3,5)
Alternaria sp., secondary leaf spot. Va. (3)
Cercospora alabamensis Atk., leaf spot. Fla. (1), Ala., N. J. (3)
C. ipomoeae Wint. Kans., Mo. (1); Ala.,Mo., Ohio (3); P. R. (5)
C. viridula Ell. & Ev. Ill., Ind., Nebr., N. J., N. Car., Tex., Va. (3)
Coleosporium ipomoeae (Schw.) Burr. (II,III), rust. N. J. to Fla., Tex., and Kans., also throughout tropical America (1,2,3,5)
Diaporthe arctii (Lasch) Nits., on stems. W. Va. (3)
Glomerella cingulata (Ston.) Spauld. & Schrenk, on stems. La. (3)
Heterodera marioni (Cornu) Goodey, root knot. Ala., Okla. (3); Hawaii (5)
Meliola clavulata Wint., black mildew. P. R. (5)
Pellicularia koleroga Cke. (Corticium stevensii Burt), thread blight. Fla. (5)
Phymatotrichum omnivorum (Shear) Dug., root rot. Tex. (1,2,3,5)
Puccinia crassipes Berk. & Curt. (I,III), rust. Fla., Ga., La., S. Car., Tex., P. R., Virgin Is. (5)
Sclerotium rolfsii Sacc., southern blight. Tex. (3)
Septoria convolvuli Desm., leaf spot. Kans. (1); Fla.,Pa., Wis. (5)
Vermicularia ipomoearum Schw., stem canker. N. Y., Pa. (3)

Mosaic -- suspected virus. P. R. (2), Fla. (5). Infection in the field and also by artificial inoculation of (3) with a strain of cucumber mosaic virus has been reported in Fla.

IPOMOEA LEPTOPHYLLA Torr., BUSH MORNING-GLORY (1); I. PANDURATA (L.) G.F.W.Mey., WILD SWEETPOTATO VINE (2); I. PES-CAPRAE (L.) Roth (3); other spp. (4). Tuberous-rooted perennials, natives of the far South and tropical America, (1) and (3) also ranging into the Eastern and Central States. All of ornamental value and sometimes cult.; (3) often becomes a persistent weed; (4) is a good soil binder.

Albugo ipomoeae-panduratae (Schw.) Swing., white rust. Colo., Kans., Tex. (1); N. J. to Fla., Ariz. and Nebr. (2); P. R., Virgin Is. (3); Guam (4)
Cercospora ipomoeae Wint., leaf spot. Fla., Ill., Miss., Mo. (2); Tex., P. R. (3)
C. viridula Ell. & Ev. Ga. (4)

Ipomoea leptophylla etc.

Coleosporium ipomoeae (Schw.) Burr. (II,III), rust. N. J. to Ala., Mo., and Kans (2); Fla., Miss., P. R. (4)
Cylindrosporium bakeri Sacc., leaf spot. Guam (4)
Heterodera marioni (Cornu) Goodey, root knot. Hawaii (3), Fla. (4)
Meliola clavulata Wint. and M. malacotricha Speg., black mildew. P. R. (4)
Phlyctaena ipomoeae Ell. & Ev., on stems and sepals. W. Va. (2)
Phyllosticta ipomoeae Ell. & Kell., leaf spot. Fla., Kans., Miss., W. Va. (2)
Phymatotrichum omnivorum (Shear) Dug., root rot. Tex. (4)
Sphaeropsis ipomoeae Ell. & Ev., on stems. W. Va. (2)

Curly top -- virus (Chlorogenus eutetticola Holmes, Beta virus 1 K.M.Sm.). Tex. (2,4)

JACQUEMONTIA (CONVOLVULACEAE)

JACQUEMONTIA SPP. Twining herbs of tropical America and the far South; sometimes grown for ornament.

Albugo ipomoeae-panduratae (Schw.) Swing., white rust. On J. tamnifolia (L.) Griseb. in Ala. and J. nodiflora (Des.) G.Don. in P. R.
Cercospora alabamensis Atk., leaf spot. Miss.
Coleosporium ipomoeae (Schw.) Burr. (II,III), rust. La., P. R. On J. tamnifolia
Heterodera marioni (Cornu) Goodey, root knot. Ala.
Uromyces gemmatus Berk. & Curt. (0,II,III), rust. P. R., Virgin Is. On J. nodiflora

QUAMOCLIT (CONVOLVULACEAE)

QUAMOCLIT COCCINEA (L.) Moench (1) and Q. PENNATA Bojer (2). Twining herbs of tropical America, grown for ornament and sparsely naturalized in the Southern States, occasionally northward; of food value to wildlife.

Albugo ipomoeae-panduratae (Schw.) Swing., white rust. N. Mex. (1), Miss. (2)
Coleosporium ipomoeae (Schw.) Burr. (II,III), rust. Ill., S. Car., Tenn., P. R., Virgin Is. (1); Miss., N. Car. (2)
Heterodera marioni (Cornu) Goodey, root knot. Ala. (1)
Phymatotrichum omnivorum (Shear) Dug., root rot. Tex. (2).
Puccinia crassipes Berk. & Curt. (I,III), rust. Virgin Is. (1)
Vermicularia ipomoearum Schw., on stems. Pa. (1)

DIVISION OF MYCOLOGY AND DISEASE SURVEY

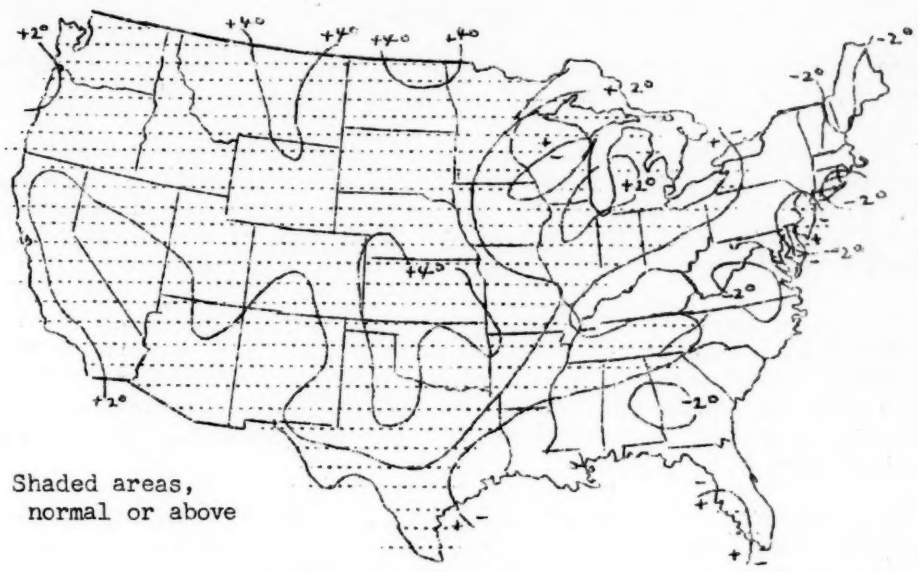
JULY WEATHER

(From U.S. Department of Commerce, Weather Bureau, Weekly Weather and Crop Bulletin for the week ending August 6, 1946)

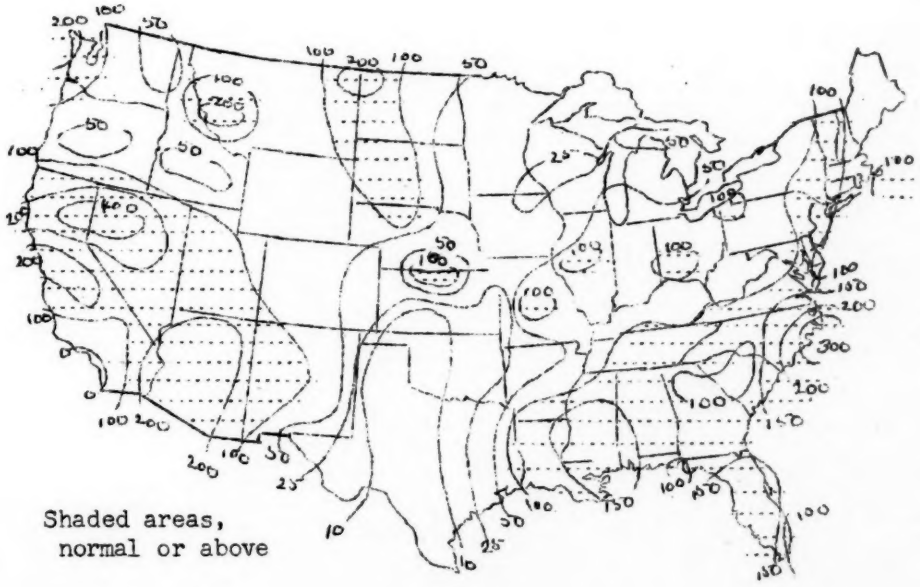
Map I shows that the month of July 1946 was warmer than usual over the western two-thirds of the country, with the plus anomalies exceeding 4° in most sections of the south-central Plains and in some northwestern areas. Temperatures averaged from mostly near normal to a few degrees below normal in the Atlantic and east Gulf States.

The rather spotted nature of the rainfall during July 1946 is evident from Map II. It was drier than usual in the greater portion of the north-central interior and the Great Plains where many areas received less than half their usual precipitation, with most stations from southern Kansas to the lower Rio Grande Valley reporting less than 10 percent of normal.

More than the usual amount of rain fell in most of the Middle Atlantic, East Gulf, and South Atlantic States and in Arizona, the Great Basin, northern and central California, western Washington, and the western Dakotas. The totals were more than twice their normals in eastern North Carolina, Arizona, northern Nevada, and sections of northern and central California.



Map I. Departure of Mean Temperature from the Normal, July, 1946.



Map II. Percentage of Normal Precipitation for July, 1946.